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CONTENTS

Volume: 3 Issue: 2 Year: 2025

ORIGINAL ARTICLE

REVIEWS

CASE REPORTS

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Performance comparison of artificial intelligence-based language models on oral pathology questions in dental education

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ABSTRACT

Aims: This study aimed to comparatively evaluate the diagnostic accuracy levels of current large language models-including ChatGPT-3.5, ChatGPT-4, Gemini, and Deepseek-through multiple-choice questions related to oral pathology, a core subject in undergraduate dental education.

Methods: A total of 30 multiple-choice questions covering oral pathology topics within the dental curriculum were prepared by a domain expert using current textbooks and course materials. These questions were applied in a text-based format to four LLMs (ChatGPT-3.5, ChatGPT-4, Gemini, Deepseek), and only the first responses were recorded without any prompting or correction. Diagnostic accuracy was assessed based on the percentage of correct answers, and inter-model agreement was evaluated using the Kappa statistic. A p-value <0.05 was considered statistically significant.

Results: ChatGPT-3.5 and ChatGPT-4.0 each achieved an accuracy rate of 93.33%, with corresponding kappa values of κ=0.951 and κ=0.927, respectively. Gemini reached 86.67% accuracy (κ=0.879), while Deepseek achieved 80% accuracy (κ=0.855). All models showed statistically significant agreement with the reference answers (p=0.000).

Conclusion: This study demonstrated that ChatGPT-3.5 and ChatGPT-4.0 outperformed other models in terms of diagnostic accuracy and agreement in answering fundamental oral pathology questions. Although all models showed significant concordance, ChatGPT-based models stood out in terms of overall accuracy. However, considering potential error margins, these models should be used only as complementary tools in dental education and should not replace primary sources of knowledge acquisition for students.

Keywords: Artificial intelligence, dentistry, oral pathology, education

INTRODUCTION

Artificial intelligence (AI) has increasingly become a prominent field, particularly through applications such as large language models (LLMs) capable of generating human-like conversations. In recent years, AI technologies have rapidly expanded across various sectors worldwide, with their areas of application growing exponentially.[1,2]

AI systems especially those based on deep learning and artificial neural networks are driving transformations in many industries, most notably in healthcare. These systems are capable of processing large datasets, analyzing relationships between concepts, and generating well-reasoned, meaningful responses.[3]

One of the most notable advancements in AI is ChatGPT (Chat Generative Pre-trained Transformer), developed by OpenAI and publicly released on November 30, 2022.[4] ChatGPT-3.5 gained recognition as the first widely accessible large language model, followed by the emergence of other advanced models such as Claude (Anthropic, USA), Gemini (Google DeepMind, USA), Copilot (Microsoft in collaboration with OpenAI, USA), and DeepSeek (DeepSeek-Vision, China).

Among these, DeepSeek, version R1 stands out as an open-source, cost-effective alternative to models like OpenAI's, offering high reasoning performance and greater accessibility. Due to its promising capabilities and affordability, it has attracted growing interest among researchers and is expected to be more widely integrated into scientific workflows.[5]

In recent years, there has been a marked increase in interest regarding the potential contributions of AI technologies in dental education, leading to a growing body of scientific research in this domain. In particular, large language models such as ChatGPT have been the focus of numerous studies examining their utility in dental education. Similarly, other LLMs including Gemini and DeepSeek have been evaluated for their potential role as complementary tools in educational contexts, with increasing representation in the literature.[6-11] However, the number of studies that comparatively evaluate ChatGPT and similar AI-based models in the context of specific curriculum components particularly clinically significant subjects such as oral pathology remains limited in the current literature.[12,13]

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Therefore, the aim of this study is to comparatively assess the diagnostic accuracy of current large language models namely ChatGPT-3.5, ChatGPT-4, Gemini, and DeepSeek using multiple choice questions related to oral pathology, a subject included in undergraduate dental curricula. This study further seeks to provide objective data on the potential applicability of these models in educational settings and to contribute a subject specific and domain focused perspective to the literature.

METHODS

Ethics Statement

This study does not involve any patient data or human participation. The questions were prepared based on course materials and were solely intended to assess knowledge level. Therefore, ethical approval was not required.[12]

Content and Topics of the Questions

In this study, a total of 30 multiple-choice questions were prepared, covering topics related to undergraduate-level oral pathology and oral mucosal lesions. The questions were designed to assess students' theoretical knowledge and were categorized under headings such as aphthous ulcers, mechanisms of white lesion formation, premalignant and malignant lesions, reactive lesions, tongue pathologies, HIV-related oral findings, histopathological concepts, infectious diseases, and pigmentation disorders related to systemic factors.

The question content was developed by a specialist in oral and maxillofacial radiology, based on current core textbooks and faculty lecture notes included in the dental curriculum. Designed with conceptual integrity that can be linked to clinical practice, the questions aimed not only to assess knowledge but also to evaluate clinical discernment, diagnostic classification skills, and critical thinking.

Testing and Comparative Evaluation of AI Models

In this study, the diagnostic performance of four advanced AI-based language models was comparatively evaluated using a standardized multiple-choice question set. All questions were presented in text-only format, and no visual content (e.g., radiographs, diagrams) was used.

The AI language models evaluated in this study were;

- ChatGPT-3.5 (OpenAI)
- ChatGPT-4 (OpenAI)
- Gemini (Google DeepMind)
- Deepseek (open-source large language model)

All models were assessed solely through text input to ensure a fair and standardized comparison.

The evaluation process was conducted as follows;

All questions were manually entered into the official web interfaces of each model by the same researcher using a single device.

Before the queries were initiated, cookies and browsing history were cleared, and all models were accessed on April 23, 2025.

Only the first response of each model was recorded, without any further input, prompting, or correction.

The responses of the models were compared against a predefined answer key, and evaluation was based solely on the accuracy of responses.

Statistical Analysis

Categorical variables were presented as frequencies (n) and percentages (%). The agreement between responses provided by different AI models was assessed using the Kappa test, and Kappa coefficients were reported. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 23.0 (IBM Corp., Released 2017). A p-value of <0.05 was considered statistically significant.

RESULTS

Following the evaluation comprising 30 multiple-choice questions, both ChatGPT-3.5 and ChatGPT-4.0 models demonstrated the highest diagnostic performance, each achieving 28 correct responses and 2 incorrect ones, corresponding to an accuracy rate of 93.33%. The Gemini model yielded 26 correct and 4 incorrect answers, with an overall accuracy of 86.67%, while the Deepseek model provided 24 correct and 6 incorrect responses, resulting in an accuracy rate of 80.00%. **Table 1** presents a comparative overview of the numerical and percentage-based distribution of correct responses across the different artificial intelligence-based language models in relation to undergraduate-level oral pathology questions.

Table 1. Frequency and percentage of predictions

Prediction tool	n	%
ChatGPT-3.5 (C/I)	28/2	93.33 /6.67
ChatGPT-4.0 (C/I)	28/2	93.33 /6.67
Gemini (C/I)	26/4	86.67/13.33
Deepseek (C/I)	24/6	80/20

C: Correct, I: Incorrect

ChatGPT-3.5 and ChatGPT-4.0 models achieved the highest performance with an accuracy rate of 93.33%. These models demonstrated statistically significant and high-level agreement, with kappa values of κ=0.951 and κ=0.927, respectively (p=0.000). The Gemini model reached an accuracy rate of 86.67% with a kappa value of κ = 0.879, indicating a high level of consistency with the reference answers (p=0.000). The Deepseek model exhibited the lowest accuracy rate at 80.00%, yet its kappa value was κ=0.855, demonstrating significant and high-level agreement with the reference responses (p=0.000). The calculated p-values for all AI models were 0.000, indicating that their agreement with the reference answers was statistically significant (**Table 2**).

Table 2. Comparison of artificial intelligence prediction models based on accuracy and consistency (kappa) metrics

Prediction tool	Accuracy (%)	Kappa value	p-value
ChatGPT-3.5	93.33	0.951	0.000
ChatGPT-4.0	93.33	0.927	0.000
Gemini	86.67	0.879	0.000
Deepseek	80.00	0.855	0.000

DISCUSSION

Although studies on the potential use of AI-based language models in health education are increasingly common,

comparative assessments targeting specific curricular subfields such as oral pathology are still limited. This study aims to contribute to the existing literature in this area.

In this context, our study demonstrated that the ChatGPT-3.5 and ChatGPT-4.o models achieved the highest accuracy rates (93.33%; κ=0.951 and 0.927; p = 0.000). Conversely, the Gemini model (86.67%; κ=0.879) and the Deepseek model (80.00%; κ=0.855) exhibited lower accuracy. Although all models showed statistically significant agreement with the reference answers, ChatGPT-based models demonstrated superior overall accuracy compared to others.

Similarly, Yılmaz et al.[12] compared eight different AI models, reporting that the ChatGPT-o.1 model achieved the highest accuracy (96%) for both knowledge-based and case-based questions. Models such as Claude 3.5, Gemini 2, and Deepseek showed significantly lower performance. These findings indicate that ChatGPT-based models perform more consistently, especially for clinical knowledge-based questions. On the other hand, another study reported that the ChatGPT-4.o model achieved the highest success, with an accuracy rate of 90.0%. In our current study, general accuracy rates were evaluated using undergraduate-level questions without clinical scenarios or specific categorization.

Additionally, Kınıkoğlu et al.,[6] analyzing data from the Dentistry Specialty Examination (DUS) for 2020-2021, found that ChatGPT-o.1 and Gemini 2.0 Advanced models achieved accuracy rates of 97.46% and 97.90%, respectively, significantly surpassing models such as ChatGPT-4.o and Gemini 1.5 Pro (p=0.0002). These findings highlight the importance of carefully evaluating performance differences among various model versions.

In contrast, Eraslan et al.,[9] in their comparative assessment within prosthodontics, found that the Copilot model had the highest accuracy (73%). This was followed by Gemini (63.5%), ChatGPT-3.5 (61.1%), Claude Pro (57.9%), and Perplexity (54.8%). Copilot significantly outperformed Perplexity (p=0.035), suggesting that model performance may vary depending on the subject area.

On the other hand, Wójcik et al.[10] reported that Claude could achieve high accuracy rates in general medical fields and maintained consistency across multilingual use; however, its moderate performance in this study indicates it may not be sufficiently optimized for specific fields such as dentistry.

Likewise, Tosun et al.[14] compared ChatGPT-3.5, ChatGPT-4, Gemini, Gemini Advanced, Claude AI, Microsoft Copilot, and Smodin AI in responding to DUS prosthodontics questions. The ChatGPT-4 model exhibited the highest accuracy (75.8%), while Gemini AI showed the lowest (46.1%). Although ChatGPT models were more successful in knowledge-based questions, all models showed limitations in case-based scenarios, suggesting the need for further optimization for complex clinical situations.

Lastly, a similar trend was observed in a study by Künz et al.[15] in restorative dentistry and endodontics. The ChatGPT-4.o model showed the highest accuracy (72%), particularly excelling in direct restoration and caries-related questions. This study also emphasized that GPT-4 and 3.5-based models provided higher accuracy levels. Additionally, another assessment with text-based case descriptions reported that the DeepSeek-R1 model reached a diagnostic accuracy of 91.6%, even surpassing ChatGPT-4.o and professional readers of the related journal.

On the other hand, Aygün et al.[11] reported high accuracy rates of AI models for anatomy questions but highlighted content inaccuracies in supplementary explanations, limiting their reliability as educational resources. Similarly, while all models evaluated in our study showed significant agreement with reference answers, incorrect or incomplete responses were observed in some questions. Thus, using these models as supplementary resources in dental education is advised, supported by supervised, multi-sourced, and critical learning approaches in fundamental knowledge acquisition processes.

Limitations

This study has several limitations. Firstly, the evaluation included only 30 multiple-choice questions, potentially restricting the assessment of general diagnostic performance of AI models. All questions were prepared in the same format (multiple-choice), excluding open-ended or case-based scenarios, limiting the ability to test clinical reasoning and problem-solving skills of the models. Additionally, as the study only covered oral pathology, findings may not be generalizable to other areas of dentistry. Finally, only initial responses from the models were evaluated, excluding interactive feedback or correction capabilities from analysis.

Future studies are recommended to include a larger number and variety of question formats, evaluate multiple dentistry fields, and examine the interactive capabilities of AI models. Such studies could provide more comprehensive assessments of model effectiveness in educational contexts.

CONCLUSION

This study revealed that ChatGPT-3.5 and ChatGPT-4.o models showed higher accuracy and statistical consistency in multiple-choice questions on fundamental oral pathology topics compared to other large language models. Although all models demonstrated significant agreement with reference responses, ChatGPT-based models exhibited superior accuracy. These results suggest these models may serve as supportive tools in dental education for specific subjects. However, considering the limitations and potential inaccuracies of AI models, their use should remain supplementary, not as primary resources for students' fundamental knowledge acquisition.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study did not require ethical approval as it did not involve any human subjects or animal experiments.

Informed Consent

Because the study has no study with human and human participants, no written informed consent form was obtained.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Review

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Diabetes and periodontitis: current approach

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ABSTRACT

Diabetes is described as a systemic disease that manifests itself with many major complications that determine the quality of life. The chronic complications of diabetes have been defined as macro- and microvascular complications. In recent years, periodontitis has also been identified as one of these complications. Diabetes negatively affects oral and dental/gum health. Metabolic disorders in diabetic patients reduce the resistance of gum tissues to infection, thereby increasing the likelihood of gum disease. Impairment of oral and dental/gum health also disrupts blood sugar regulation in diabetic patients. Thus, the negative effect is two-way. The relationship between periodontitis and diabetes has also been observed in many studies. The aim of this study is to provide information about diabetes and periodontitis, explain the relationship between diabetes and periodontitis, and offer the best possible dental care and counseling to diabetic patients based on general dental knowledge.

Keywords: Diabetes, oral health, dental health, periodontitis

DIABETES MELLITUS

The guideline prepared by the Society of Endocrinology and Metabolism of Turkiye (SEMT) and frequently referred to in the follow-up and treatment of diabetes patients in our country defines diabetes mellitus (DM) as a chronic, broad-spectrum metabolic disorder that requires ongoing medical care.[1] Characterized by hyperglycemia, DM, according to the American Diabetes Association (ADA), arises due to hyperglycemia caused by insulin deficiency or defects in insulin action.[2]

Diabetes Diagnosis Criteria

- According to the current SEMT guidelines, one of the four diagnostic criteria is sufficient for a diagnosis of DM (SEMT, 2024).[1] These four diagnostic criteria are as follows:
- Fasting plasma glucose (FPG) level ≥126 mg/dl after ≥8 hours of fasting
- Oral glucose tolerance test (OGTT) 2-hour plasma glucose (PG) level ≥200 mg/dl
- The glycated hemoglobin A1c (HbA1c) level ≥6.5%.

If one of these three criteria is met, it must be confirmed by another criterion.

- Random plasma glucose level ≥200 mg/dl accompanied by diabetes symptoms (such as excessive eating, excessive thirst, and frequent urination). If this criterion is present, a diagnosis can be made.

Epidemiology

DM is a chronic health problem that has reached alarming levels worldwide. According to data from the International Diabetes Federation (IDF), in 2019, the estimated number of people with DM worldwide reached 463 million (9.3% prevalence) and resulted in 4.2 million deaths. It is projected that the prevalence of DM will rise to 9.9% by 2045, reaching 693 million people. In Turkiye, the TURDEP-II study conducted between 2009 and 2010 found that the prevalence of DM was 16.5%, with 6.5 million adults diagnosed with DM. Today, it is estimated that one in three patients has diabetes/prediabetes.[1-4]

Complications of Diabetes

The General Directorate of Health Services published the Turkiye Diabetes Prevention and Control Program Action Plan (2011-2014) as part of the Ministry of Health of the Republic of Turkiye's 2010-2014 Strategic Plan, which includes the sub-goal of "ensuring early diagnosis and treatment of diabetes and reducing the incidence of diabetes-related complications by the end of 2014." The aim of this plan is to ensure the early diagnosis and treatment of diabetes, raise public awareness of related risk factors, and reduce the incidence of diabetes-related complications to World Health Organization targets. Within the plan, the objective of "improving the quality of life of people with diabetes" is listed as the first activity under the target of "developing care services for people with diabetes," specifically "ensuring regular monitoring of oral and dental health for people with diabetes."[5-7]

Diabetes leads to many chronic complications.[1,6,7]

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Acute Complications of Diabetes

Acute complications of diabetes mellitus are classified under four main headings.

- Diabetic ketoacidosis
- Hyperosmolar hyperglycemic state
- Lactic acidosis
- Hypoglycemia

Chronic Complications of Diabetes

- **Macrovascular complications:** Atherosclerotic heart disease, cerebrovascular disease, and peripheral vascular disease
- **Microvascular complications:** Retinopathy, nephropathy, neuropathy

In recent years, periodontal diseases have also been considered one of the chronic complications of diabetes.[8-10]

PERIODONTAL MEDICINE

First published in 1891, the focal infection theory demonstrated that oral microorganisms and/or their products could reach other parts of the body (Miller, 1891).[11] Later, it was suggested that infected teeth could be a source of focal infection in certain diseases such as arthritis, rheumatism, nephritis, and endocarditis (Billings, 1914).[12] Those who supported this theory argued that dental plaque microorganisms and their metabolic products could enter the bloodstream and cause many systemic and some degenerative conditions. In the 1930s, it was observed that patients' systemic conditions did not improve even after the suspected source of focal infection was eliminated, and this theory began to be questioned. After 1990, the relationship between oral/periodontal infection and systemic health was investigated under the name "periodontal medicine." Periodontal medicine is defined as a discipline that investigates the two-way relationship between systemic health and periodontal disease and the possible mechanisms involved in this relationship.[11-13]

Gram-negative bacteria found in the subgingival area of individuals with periodontal disease are a constant and significant source of irritation for the host. Periodontal bacteria and their products (such as lipopolysaccharides) can mostly enter periodontal tissues and the circulation through ulcerated and disrupted sulcular epithelium. Inflammatory markers such as TNF-α, IL-1ß, prostaglandin E2 (PGE2), and γ-interferon are produced in inflamed gingival tissue; these can enter the bloodstream and contribute to the inflammatory load. Thus, while periodontal tissues mount an immune-inflammatory response to bacteria and their products, the systemic effects of these agents also result in a major vascular response. This may explain the relationship between periodontal disease and systemic diseases. Three mechanisms by which periodontal infection may affect systemic health have been identified;[10,13,14]

- Metastatic infection caused by translocation of gram-negative bacteria from the periodontal pocket into the bloodstream
- Metastatic injury such as vascular lesions caused by the effects of circulating microbial toxins and proinflammatory markers
- Metastatic inflammation associated with the immune response to periodontal pathogens and their products

In recent years, periodontal inflammation has been identified as a risk factor for the development and progression of various systemic diseases and conditions. The relationship between periodontal disease and DM, cardiovascular disease, chronic obstructive pulmonary disease, and adverse pregnancy outcomes (low birth weight babies and premature birth), as well as its relationship with diseases and conditions such as obesity, metabolic syndrome, rheumatoid arthritis, chronic kidney disease, and cancer, are being investigated, along with the possible mechanisms involved.[15-19]

THE RELATIONSHIP BETWEEN DIABETES MELLITUS AND PERIODONTITIS

There is a bidirectional relationship between DM and periodontal diseases due to DM being a risk factor for periodontal diseases because it increases the prevalence and severity of periodontitis, and periodontitis making glycemic control difficult.[9,10]

Both periodontal disease and T2DM have been reported to have genetic factors. Periodontitis has been reported to be a complication of DM. DM is a major risk factor for periodontitis. It has been shown that the severity and prevalence of periodontal disease increase in individuals with DM. Metabolic control of DM may be an important variable in the onset and progression of periodontal disease. Many studies have found that the risk of developing periodontal disease is much higher in individuals with DM compared to those without DM. Improvements in metabolic status in individuals with T2DM positively affect periodontal health, while metabolic control is positively affected in individuals with DM who undergo periodontal treatment. Bleeding after periodontal procedures decreases with improved glycemic control. Most comprehensive meta-analyses have concluded that adults with DM have more severe periodontal disease compared to adults without DM. DM has been associated with an increased risk of periodontitis even at a young age.[10,15-19]

The Effect of Diabetes Mellitus on Periodontal Disease

Many of the mechanisms involved are similar to those of microvascular and macrovascular complications. In addition, hyperglycemia observed in DM may cause the activation of pathways that increase inflammation, oxidative stress, and apoptosis. Structural components such as proteins, lipids, and nucleic acids in our body undergo glycation with monosaccharides such as fructose, galactose, and glucose. Glycation, an irreversible process, occurs spontaneously and results in the formation of advanced glycation end products (AGEs). AGEs accumulate in cells and tissues, forming cross-links with other protein and lipid molecules, which leads to the inactivation of structures and functional impairment. The receptors targeted by AGEs in structures such as smooth muscle cells, endothelial cells, monocytes, and fibroblasts are called RAGE (receptor for AGE) and exert their effects by forming a dimer structure called AGE-RAGE. RAGE is synthesized in small amounts in healthy individuals but increases in individuals with DM. Hyperglycemia triggers a series of immune responses through the production of AGEs. The binding of AGEs to their receptors leads to increased production of inflammatory markers such as IL-1ß, TNF-α, and IL-6. AGE formation increases oxidative stress by causing the production of reactive oxygen species, and the resulting

endothelial cell changes contribute to vascular damage, which plays a role in many diabetic complications. AGEs exhibit harmful effects on bone metabolism by causing impaired tissue repair and bone formation and reduced extracellular matrix production. In conclusion, hyperglycemia causes AGEs to accumulate in periodontal tissues and bind to the AGE receptor, leading to the release of local cytokines and subsequent changes in inflammatory responses. As the inflammatory response persists, an increase in periodontal and bone destruction is observed. In DM, neutrophil chemotaxis and phagocytosis are impaired, and bacterial retention, which causes destruction of periodontal tissues, is increased. In DM, although neutrophil function is reduced, an increase in DOS monocyte and macrophage counts is observed. This increase leads to elevated levels of pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6, contributing to increased periodontal tissue destruction in individuals with DM. The abnormal host defense observed in individuals with DM, in addition to hyperglycemic status, contributes to the proliferation of periodontopathogens in gingival crevicular fluid (GCF) and saliva. With increasing severity of DM, an increase in periodontal abscess formation and tooth loss due to periodontal destruction is observed. DM also has a significant effect on collagen metabolism; in the presence of hyperglycemia, the maturation and homeostatic turnover of collagen produced by fibroblasts is impaired, resulting in a decrease in the amount of newly produced collagen. The newly formed collagen is not very strong and is highly susceptible to degradation by certain matrix metalloproteinases, such as collagenase. Existing collagen forms structures that cannot be dissolved by AGEs, leading to collagen imbalance and delayed, inadequate tissue healing in periodontal disease.[17,18,20]

The Effect of Periodontitis on Diabetes Mellitus

Periodontitis is characterized by the infiltration of inflammatory cells into periodontal tissues. It has been shown that the increased inflammatory cytokines that occur in periodontal disease cause tissue destruction and have systemic effects. It is thought that the presence of periodontal inflammation in DM patients makes glycemic control more difficult. Chronic gram-negative periodontal infections may cause increased insulin resistance and poor glycemic control. Studies have shown that in periodontitis patients, serum inflammatory markers such as gram-negative organisms, CRP, IL-6, and fibrinogen are significantly higher. The systemic spread of these organisms or their products may trigger bacteremia or endotoxemia, a high inflammatory state, and an increase in serum inflammatory markers. Severe periodontal inflammation further increases circulating TNF-α concentrations by stimulating monocytes, which can have additional effects on insulin resistance by directly affecting target organs such as the liver, muscles, and adipocytes, and indirectly increasing the release of other insulin resistance molecules such as free fatty acids from adipocytes. In diabetic patients with severe periodontitis, the mortality rate due to ischemic heart disease was found to be higher than in diabetic patients without periodontitis or with mild periodontitis. When other known risks were taken into account, the mortality rate due to diabetic nephropathy was 8.5 times higher in DM patients with severe periodontitis. In a study evaluating over 600 patients with T2DM, it was demonstrated that the mortality rate due to cardio-renal disease was 3.5 times higher in patients with severe periodontitis.[17,21,22]

In a long-term case-control study, 82% of patients with DM and severe periodontitis and 21% of patients with DM and no periodontitis were found to have one or more major cardiovascular, peripheral vascular, and cerebrovascular events.[23]

PERIODONTAL HEALTH, GINGIVAL DISEASES AND CONDITIONS

Periodontal Health

Periodontal health is defined clinically as the absence of inflammatory periodontal disease. Periodontal disease includes both gingivitis and periodontitis. Gingivitis occurs as a result of the inflammatory response, which is the host's first defense mechanism against bacterial dental plaque that accumulates on the tooth surface. If this inflammatory response continues and is not treated, the lesion progresses to deeper tissues and causes loss of alveolar bone, which leads to periodontitis. Periodontitis is a disease that destroys the supporting structures of the teeth, including the periodontal ligament, bone, and soft tissues. Periodontal diseases, which are usually painless, are often noticed by the patient only in their late, irreversible stages.[14,16,24]

Classification of Periodontal Diseases

Periodontal diseases have been classified in many ways to date. The most recent classification was developed in 2017 by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) following a meeting attended by experts. The classification includes the pathogenesis of periodontal conditions and diseases, their multifactorial etiology, the causes of the disease, and peri-implant diseases.[24]

According to the AAP and EFP, periodontal and peri-implant diseases and conditions were classified as follows in 2017 (**Table 1**).[24]

Periodontal Health, Gingival Diseases and Conditions

Gingivitis (induced by dental biofilm): Gingivitis is an inflammatory disease of the gums that occurs when the balance between bacteria colonizing the gingival sulcus and the host response is disrupted. Radiologically, bone loss is not observed in gingivitis. The clinical signs of gingivitis include changes in gum color, plaque on the gum line, increased DOS, bleeding on probing, changes in gum pocket temperature, changes in gum contour, and no attachment loss. Bleeding on probing and increased DOS are the earliest clinical signs of gingivitis. Gingivitis can be eliminated by removing plaque.[23-25]

Gingival diseases (not induced by dental biofilm): It covers a range of conditions that are not caused by bacterial dental plaque and therefore do not improve with plaque removal (Tonetti et al., 2018). Gingival diseases and conditions not induced by dental plaque/biofilm were classified as follows by the AAP and EFP in 2017.[23-26]

- Genetic/developmental disorders
- Specific infections
- Inflammatory and immune conditions
- Reactive processes
- Neoplasms
- Endocrine, nutritional, and metabolic diseases
- Traumatic lesions
- Gingival pigmentation

Table 1. Overview of periodontal diseases and conditions			
Periodontal diseases and conditions			
Periodontal health, gingival diseases and conditions	Periodontal and gingival health		
	Gingivitis (induced by dental biofilm)		
	Gingival diseases (not induced by dental biofilm)		
Periodontitis	Necrotizing periodontal diseases		
	Periodontitis		
	Periodontitis associated with systemic diseases		
Other conditions affecting the periodontium	Systemic diseases and conditions affecting periodontal support tissues		
	Periodontal abscesses and endodontic-periodontal lesions		
	Mucogingival deformities and conditions		
	Traumatic occlusal forces		
	Dental and prosthetic factors		
Peri-implant diseases and conditions			
Peri-implant health	Peri-implant mucositis	Peri-implantitis	Peri-implant soft and hard tissue deficiencies

PERIODONTITIS

Periodontitis is an inflammatory disease characterized by deep pockets and bone loss, associated with apical migration or attachment loss of the epithelium along the root surface due to microbial plaque accumulation. Periodontitis follows gingivitis and is also influenced by the host's immune and inflammatory response. Periodontitis is characterized by the destruction of the supporting structures of the teeth, including the periodontal ligament, bone, and soft tissues. In some cases, exposure of the root surface, gingival overgrowth or recession, furcation involvement, mobility, and the presence of pus may also be observed.[22-26] It has been demonstrated that preventing plaque accumulation through various professional and home-based techniques is highly effective in preventing attachment loss over a 15-year period.[27]

Periodontitis Stages

It can be categorized into four different groups in relation to the severity of the disease and the complexity of its treatment. In staging, factors such as loss of clinical attachment, tooth loss due to periodontal causes, percentage of bone loss, furcation involvement, angular defects are taken into consideration.[16,26]

Stage I periodontitis: There is a maximum of 1-2 mm interproximal clinical attachment loss. There is no tooth loss due to periodontal disease. There is less than 15% radiographic bone loss in the coronal triad. There is a pocket depth of 4 mm or less on probing.

Stage II periodontitis: There is a maximum of 3-4 mm interproximal clinical attachment loss. No tooth loss due to periodontal disease is observed. Radiographic bone loss in the coronal triad is between 15-33%. There is a pocket depth of 5 mm or less on probing.

Stage III periodontitis: Interproximal clinical attachment loss is 5 mm or more. Maximum tooth loss due to periodontal disease is 4 teeth. Radiographic bone loss extending to the middle or apical third of the root is observed. There is a pocket depth of 6 mm or more on probing. Deep intraosseous defects, furcation involvement, moderate crest defects and tooth loss make the treatment complex. Chewing function is preserved at this stage.

Stage IV periodontitis: Interproximal attachment loss is 5 mm or more. Radiographic bone loss extending to the middle or apical third of the root is observed. Class 2 or more mobility and masticatory dysfunction may be observed. There is loss of 5 or more teeth due to periodontitis and the number of remaining teeth is less than 20. Chewing function is impaired.

Degrees of Periodontitis

It grades the patient's individual susceptibility to periodontitis. Bone loss/age ratio is an important parameter used to determine the extent of the disease. C-reactive protein (CRP), HbA1c and smoking are risk factors that affect the rate of progression of the disease and may accelerate the transition to the next stage (**Table 2**).[26] Grading helps in diagnosis and treatment as it includes individual patient factors.[24-27]

PERIODONTAL PATHOGENESIS

More than 108 species have been isolated from 1 mm3 of dental plaque weighing approximately 1 mg. Approximately 800 different bacterial species have been identified in the oral cavity and 50-60 different species have been isolated from dental plaque samples. Pathogenic microorganisms in plaque and their products initiate the inflammatory response. As a result of the interaction between these etiologic agents in dental plaque and the host tissue response, destruction of periodontal tissues is observed. Microorganisms in dental plaque must have at least 3 characteristics in order to be pathogenic;[22-28]

- Colonization of periodontal tissues
- Overcome host antibacterial defense mechanisms
- Secreting substances that can cause tissue destruction

According to the histopathologic characteristics of these changes, 4 stages of periodontal disease progression have been defined as initial, early, established and advanced lesions.[22-28]

Initial Lesion

It can be defined as the physiologic response of clinically healthy gingiva without signs of inflammation to a small amount of microbial dental plaque. It occurs in the first 4 days following plaque accumulation and changes in the tissues

Table 2. Grades of periodontitis

Grades of periodontitis			Grade A: slow rate of progression	Grade B: moderate rate of progression	Grade C: rapid rate of progression
Primary criteria	Evidence of direct progression	Longitudinal data (radiographic bone loss or CAL)	No bone loss over 5 years	More than 5 years <2 mm	More than 5 years ≥2 mm
	Evidence of indirect progression	% bone loss/age	<0.25	0.25-1.0	>1.0
		Case phenotype	Low level of degradation relative to the amount of biofilm	Degradation proportional to biofilm	Greater degradation relative to the current biofilm: Specific clinical patterns suggestive of periods of rapid progression and/or early disease (e.g., molar/incisor pattern; lack of expected response to standard bacterial control therapies)
Degree modifiers	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥ 10 cigarettes/day
		Diabetes	Normoglycemic/no diagnosis of diabetes	HbA1c <7.0 % diabetes patient	HbA1c ≥7.0 % diabetes patient
Risk of systemic effects of periodontitis	Systemic load	High sensitivity CRP (hsCRP)	<1 mg/L	mg/L	>3 mg/L
Biomarkers	CAL/indicator of bone loss	Saliva, gingival crevicular fluid, serum	?	?	?

CAL: Clinical attachment loss, CRP: C-reactive protein, hsCRP: High sensitive CRP

are only visible histologically. There are increased spaces between endothelial cells, vasodilatation in the vasculature and high hydrostatic pressure in the microcirculation. These changes lead to increased vascular permeability and allow the migration of neutrophils and monocytes towards the bacterial products in the gingival sulcus causing a chemotactic effect. The increase in DOS helps to wash away bacteria and their products by dilution of the bacterial products.[26]

Early Lesion

It covers the period of 4-7 days following plaque accumulation and early clinical findings are observed. It develops as a result of the failure of the host immune response to eliminate the infection. The gingiva is erythematous as a result of vasodilatation. Increased vascular permeability increases the flow of DOS. There is an increase in macrophages, neutrophils, lymphocytes, mast cells and plasma cells in the connective tissue. 75% of these lymphocytes are T cells. Fibroblasts degenerate through apoptosis and collagen fibers begin to break down to provide space for leukocytes. Collagen tissue destruction is increased by 60-70% and retepegs are present in the junctional epithelium. There is redness and edema of the gingival tissues and bleeding on probing.[26-28]

Localized Lesion

It is the stage that occurs between 14-21 days following plaque accumulation, in which inflammation cannot be stopped and is called "chronic gingivitis". As inflammation continues, connective tissue destruction and bone tissue damage increase. Along with macrophages, T and B lymphocytes and plasma cells are dominant in this stage. Pocket drainage is impaired as a result of more apical migration of the junctional epithelium. With increased vascular permeability, inflammatory cytokines; inflammatory markers such as IL-1, TNF- α, PGE2 continue tissue destruction. While the inflammatory cell infiltrate settles in deep tissues, collagen destruction continues with collagenase activity. The junctional epithelium transforms into a pocket epithelium that allows the biofilm to migrate into deeper tissues. Bone destruction is not yet seen at this stage. Moderate inflammation is seen clinically in the gingiva.[26-28]

Progressed Lesion

It is the stage of transition from gingivitis to periodontitis in which destruction of periodontal tissues begins. Loss of attachment can be observed both histologically and clinically. Inflammatory cell infiltration with a predominance of plasma cells penetrates deeper tissues. Inflammatory cytokines (IL-1, PGE2 and TNF-α) stimulate fibroblasts to produce matrix metalloproteinase which causes extracellular matrix degradation. As the junctional epithelium migrates apically, collagen degradation increases. Attachment loss and bone loss become visible at this stage. Not all gingivitis lesions progress to periodontitis. The effects of bacteria on host tissue, host immune response, genetic and environmental factors play a role in the progression to periodontitis. The determining factor in bone loss is neutrophil count. Neutrophils are an important source of IL-17, an osteoclastic cytokine. They can induce inflammatory cells such as lymphocytes, polymorphonuclear leukocytes and macrophages. Lymphocytes can activate macrophages to synthesize and secrete a number of molecules such as IL-1 and TNF-α-like cytokines, proteolytic enzymes and PGE2. Lymphotoxin, a molecule similar to TNF-α and IL-1, is produced when bacterial agents activate T lymphocytes. These cytokines play a key role in periodontal tissue destruction. Metalloproteinase products cause extracellular tissue destruction. Both TNF-α and IL-1 production are suppressed by PGE2, exerting a control on the development of periodontal disease. Transforming growth factor-ß (TGF-ß)-like cytokines suppress matrix metalloproteinase production. The progression and extent of tissue destruction can be determined by the activity of molecules such as TNF-α, IL-1, PGE2 and TGF-ß.[26-30]

PERIODONTAL TREATMENT

The aim of periodontal treatments is to stop tissue destruction and disease progression by relieving inflammation in periodontal tissues. Initial periodontal treatment includes comprehensive teaching of plaque control, complete removal of calculus and bacterial dental plaque, treatment of existing decayed teeth and treatment of inappropriate restorations. Improvements in pocket depth, gingival index, clinical attachment level and bleeding on probing have been shown

after initial periodontal treatment. Periodontal treatment is divided into non-surgical and surgical treatment. Removal of microbial dental plaque accumulated in the supragingival and subgingival areas is the primary procedure for the removal of inflammation in periodontal tissues. Non-surgical periodontal treatment is a periodontal treatment that includes scaling and root surface smoothing. In non-surgical periodontal treatment, manual crettons and curettes or ultrasonic instruments or a combination thereof are used. Both types of instrumentation have similarly improved clinical parameters such as decreased pocket depth, decreased bleeding at probing and decreased clinical attachment loss. Non-surgical periodontal treatment of patients with advanced periodontitis with good oral hygiene has been shown to yield similar results when performed alone and in combination with the modified Widman flap. In a study comparing the mean of clinical measurements, no difference was observed between non-surgical and surgical treatments and non-surgical treatment was shown to be an effective treatment. Following nonsurgical periodontal treatment, clinical attachment gains and significant reduction in pocket depth were observed in areas with pocket depths of 4-6 mm and ≥7 mm.[3,13,16,26-32]

Lipotoxicity is a critical factor that not only aggravates the complications of diabetes, but also increases the severity and progression of diabetes-related periodontitis. Treatment approaches targeting this process are becoming increasingly important. Thanks to the lipotoxicity-reducing effects of agents such as metformin, statins, liraglutide, adiponectin and omega-3 fatty acids, it is thought that periodontal tissue damage can be reduced by controlling processes such as oxidative stress, inflammation and cell death. Accordingly, treatment strategies targeting lipid metabolism are promising in the management of diabetes-related periodontal diseases.[33]

He examined the effect of SIRT3 protein on the senescence and bone-forming ability of human periodontal ligament stem cells in diabetes-induced periodontitis. SIRT3 was shown to be suppressed under diabetic conditions, which accelerated cell senescence and inhibited bone regeneration. SIRT3 was also found to regulate this process by acetylating a protein called LRPPRC, and a compound called honokiol activated SIRT3 to delay aging and promote bone healing. The results revealed that SIRT3 may be a novel therapeutic target for diabetes-associated periodontitis (SIRT3 is a next generation biomolecular therapeutic agent that can be targeted to protect periodontal tissues and promote regeneration in diabetic conditions).[34]

It emphasizes that immune system imbalance plays a fundamental role in the development of diabetes-associated periodontitis (DPD). There is limited but promising evidence that some existing drugs (e.g. metformin, antibiotics, vitamin D) may provide potential benefit in the treatment of DPD by modulating the immune response. Therefore, drug therapies targeting the immune system are a promising approach for DPD.[35]

This study proposes hydrogel microneedle systems enriched with biologically active glass nanoparticles containing zinc and vanadium for the treatment of diabetic periodontitis. These microneedles exhibit antioxidant and antibacterial properties, providing direct drug delivery to gingival tissue and suppressing inflammation. Experiments in animal models have shown that this method enhances alveolar bone regeneration and suppresses inflammatory signaling pathways (JAK-STAT, NF-κB).[36]

This study revealed common genetic and immunological pathways between T2D and periodontitis, identifying the NCF1 and LRRC25 genes as new therapeutic targets for these two diseases. Furthermore, cellular function and metabolic pathways were shown to be altered depending on whether monocytes express these genes or not, thus biologically supporting the observed causal relationships.[37]

CONCLUSION

Diabetes is a systemic disease characterized by many major complications that determine the quality of life. Periodontitis is one of these complications. The metabolic disorders of patients with diabetes reduce the resistance of gingival tissues against infection, and the occurrence of gingival disease comes to the fore. The relationship between periodontitis and diabetes has been observed in many studies. The aim of this study is to provide information about diabetes and periodontitis, to explain the relationship between diabetes and periodontitis, and to provide the best possible dental care and counseling to the diabetic patient with general dental knowledge.

ETHICAL DECLARATIONS

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Periodontal risk

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ABSTRACT

Periodontal diseases are chronic inflammatory conditions primarily initiated by microbial biofilms. However, not all individuals with similar plaque accumulation develop periodontitis, indicating the influence of various risk factors. This review aims to summarize the evidence-based general and individual risk factors, indicators, and predictors associated with periodontal disease. General risk factors include microbial pathogens, poor oral hygiene, smoking, and diabetes mellitus. Individual risk factors encompass non-modifiable elements such as genetic predisposition, age, gender, socioeconomic status, and psychological stress. Additionally, conditions like HIV/AIDS, osteoporosis, and irregular dental visits are identified as risk indicators, while clinical findings such as bleeding on probing and history of periodontitis serve as risk predictors. Understanding and evaluating these factors are crucial for preventive strategies, disease management, and improving periodontal prognosis.

Keywords: Periodontal disease, risk factors, risk indicators, risk predictors

STRENGTH OF EVIDENCE FOR RISK DEFINITION AND RISK FACTORS

The word "risk" originates from French, and its root word is "riziko." The dictionary definition of "riziko" is the danger of suffering loss.[1]

In health research, risk is defined as the likelihood of a person developing a specific disease over time. Risk is the probability of an event occurring. In epidemiology, it is often used to indicate the probability of a specific event occurring.[2] In chronic diseases and infections, the same outcome is not always achieved; there are very few situations that constitute sufficient cause.[3]

The impact of each scientific study on epidemiological evidence is determined not only by the quality of the study but also by the design and methods used, which determine what conclusions about causality can be drawn from the data obtained. In human health, a causal factor is defined as a factor whose presence increases the occurrence of a disease or condition.[4]

To establish causality, certain criteria must be met, such as the presence of the cause before the disease outcome (temporality) and biological plausibility.[5]

Potential risk factors can be identified through data obtained from epidemiological studies for use in subsequent advanced studies. These indicators then provide evidence that sheds light on how etiology and risk factors work at the cellular, genetic, and molecular levels.[6]

After reviewing the evidence, recommendations can be made for modifying risk factors for preventive measures in public health.[4]

One of the most complex issues in epidemiological studies is determining causal inferences. In 1971, Hill defined the criteria that must be met to see causal inferences:[7]

Strength of the Relationship

The stronger the relationship between a potential risk factor and the occurrence of a disease, the more valid the expected causal relationship. As the strength of the relationship increases, the likelihood of causation increases; estimated relative risk and relative risk confirm this strength.

Dose-Response Effect

If the frequency of the disease increases as the dose or level of exposure to a factor increases, this supports a causal relationship. An increase in the factor's presence and dose is expected to lead to an increase in the disease.

Temporal Consistency

It is important to determine whether exposure to the suspected factor occurred before the disease developed. This may be difficult to determine in diseases with a long latent period or where factors may change over time.

Consistency of Findings

Another criterion that strengthens causal interpretation is the obtaining of similar results in multiple studies examining a specific relationship.

Biological Plausibility

The expected relationship should be plausible when evaluated in light of existing biological knowledge. However, it should

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be noted that when data on the etiology of a particular disease are limited, this criterion may be more difficult to meet.

Specificity of the Relationship

If the disease is found to be associated with only one of the factors under investigation, or if the factors under investigation are associated with only one disease, the causal relationship is strengthened.

GENERAL RISK FACTORS

Periodontal disease is an infectious bacterial disease caused by dental plaque. The virulence of the main pathogens found in the subgingival biofilm can influence the etiology and pathogenesis of the disease. In the 1960s, people thought everyone was equally likely to get periodontal disease, but later studies using measurements like pocket depth, attachment loss, and gingival inflammation showed that only some people experience severe periodontitis, indicating that certain individuals are more at risk for the disease. This has led to the conclusion that some individuals are more susceptible to periodontal disease.[9]

Risk factors were defined by Beck[10] in the 1996 World Journal of Periodontology. They are defined as environmental, behavioral, or biological factors that, when present, directly increase the likelihood of disease onset, and when absent or eliminated, substantially reduce the likelihood of disease onset, as identified in longitudinal studies with a temporal sequence.

Behavioral, environmental, or biological factors that increase a person's likelihood of developing a disease are risk factors. These factors are links in the causal chain and are directly related to the occurrence of the disease. These factors are identified through long-term studies of individuals who have experienced the disease in question. Exposure to a risk factor may occur within a specific time frame, may occur multiple times at different times, or may be continuous over a long period of time. For a factor to be defined as a risk factor, exposure to the factor must occur before the onset of the disease.[11]

Pathogenic Bacteria and Adjuvants

Periodontitis is a chronic inflammatory disease that can cause progressive destruction of the tissues supporting the teeth if left untreated.[12] The accumulation of microbial plaque and the host's immune and inflammatory responses to enzymes such as nucleases, lipases, and proteases are the primary etiological factors of periodontal disease.[13] One of the most heterogeneous microbial communities found in humans is the oral microbiota, which ranks second after the gastrointestinal microbiota in terms of complexity and species diversity.[14] The complexity is due to the different environmental conditions in different regions of the oral cavity. Different environmental conditions (gingival sulcus, tongue, teeth, lips, hard and soft palate, buccal mucosa, keratinized gingiva, and non-keratinized gingiva) provide distinct habitats for microbial colonization. This allows microorganisms to obtain suitable environments for biofilm formation.[15]

The host's initial response to periodontal disease is an acute inflammation against supragingival and subgingival plaque, managed by components of the innate immune system such as epithelial cells, macrophages, neutrophils, fibroblasts, complement proteins, and neuropeptides. At this stage, cytokines such as interleukin-1β, interleukin-6, and tumor necrosis factor-α, produced by resident cells, increase the expression of adhesion molecules for neutrophils on the inner surface of blood vessels, cell migration to the infection site, and the synthesis of other pro-inflammatory cytokines.[16]

The elimination of the plaque contributes to the gradual resolution of inflammation and the reestablishment of individual homeostasis. The continued presence of the plaque triggers the activation of the acquired immune response. Antigens are processed and presented by lymphocytes, dendritic cells, and macrophages. These processes are regulated by acquired immune cytokines such as interferon (IFN)-γ and interleukin-2 and interleukin-4. Inflammation and cytokines disrupt the balance between osteogenesis and osteoclastogenesis in bone tissue, shifting it toward osteoclastogenesis and causing destruction.[17]

Axelsson et al.[18] conducted a study involving 375 participants over a 15-year period. All participants received oral hygiene education, and tartar removal and root planing were performed every two or three months for nine years, followed by twice a year for the next six years. The results indicated that no clinically detectable attachment loss was observed in any of the participants.

In individuals who regularly maintained oral hygiene, a certain amount of dental calculus did not cause significant attachment loss, whereas in those who did not maintain regular oral hygiene, the presence of dental calculus increased the likelihood of attachment loss by fivefold. The reported associations between dental calculus, plaque, and periodontal tissues suggest that these factors may serve as risk markers for periodontal disease.[19]

Abdellatif and Brut,[20] in their study involving 14,690 individuals aged 15 to 74, found a 20.52% association between periodontitis and poor oral hygiene. In all age groups in the study, the incidence of periodontitis was significantly higher in individuals with poor oral hygiene. Hansen et al.,[21] in their study including individuals aged 50 and older, found a 10.86% association between an OHI-S score of 2.0 or higher and pocket formation.

Restorations and prostheses may influence biofilm development and formation due to their unique chemical and physical properties. Rough surfaces contain irregular areas that are ideal for colonization and plaque formation.[22]

Smoking

When the contents of a cigarette are examined, there are over 6,000 substances that are cytotoxic, carcinogenic, and mutagenic in gaseous or particulate form. Cigarettes contain toxins such as hydrogen cyanide, ammonia, formic acid, ketones, and nitrites, and carcinogenic substances such as nickel, chromium, arsenic, benzopyrene, formaldehyde, and acetaldehyde. Each cigarette consumed contains approximately 20-30 ml of carbon monoxide and 2-3 mg of nicotine.[23]

Cigarette consumption is one of the well-known risk factors for periodontal disease. Numerous studies have demonstrated a relationship between cigarette consumption and the destruction of periodontal tissues.[24] It has been reported that smoking increases the risk of periodontal attachment or bone

loss by 2 to 8 times, depending on the stage of periodontal disease and the amount consumed.[25]

A meta-analysis including six randomized controlled trials found that severe periodontitis was three times more prevalent in smokers than in non-smokers.[26] Studies comparing smokers and non-smokers with similar plaque accumulation found that those who smoked had greater probing depth, attachment loss, bone loss, and tooth loss.[27] It has been determined that the consumption dose of smoking affects this risk and that the risk increases with increased consumption.[28]

The effect of smoking on non-surgical and phase 2 periodontal treatment: Studies have shown that individuals who smoke after undergoing non-surgical periodontal treatment, hard tissue surgery, or soft tissue surgery experience less clinical attachment gain. It has been reported that smoking delays the healing process after surgical interventions by inhibiting revascularization and suppressing growth factors.[29]

Kinane et al.[30] investigated the effects of smoking on 54 patients following antimicrobial and mechanical treatment. In all groups, they found greater attachment gain and changes in pocket depth in non-smokers.

Preber et al.,[31] in their study examining pocket depth improvement, observed lower healing in the maxillary anterior teeth of smokers following treatment. Although the exact cause is unknown, they suggested that plaque differences, greater exposure of the area to smoking compared to other regions, or a combination of these factors may be responsible.

Smoking and gingivitis: Bleeding on probing is the earliest sign of gingivitis and is used to clinically identify active disease. Smoking causes vasoconstriction in peripheral blood vessels, reducing blood flow to periodontal tissues, which results in fewer signs of gingivitis in smokers compared to non-smokers. In cases of simple gingivitis, no clinically significant difference is observed between smokers and non-smokers in terms of gum appearance. However, in studies using methods such as bleeding on probing and gingival index, it has been observed that inflammatory reactions are more prominently masked in smokers when plaque levels are high. Therefore, it has been reported that severe gingivitis is not commonly observed in smokers.[32]

Smoking and periodontitis: In clinical studies standardized for age, race, and education level, it has been reported that smokers are 3-4 times more likely to develop periodontitis and that bone loss increases in proportion to the number of cigarettes smoked per year.[33]

In a study examining 12,329 American adults aged 20 and older, it was found that current smokers were four times more likely to have periodontitis than non-smokers. The same study also reported that heavy smokers had a higher risk ratio than light smokers.[34]

Smoking and acute necrotizing ulcerative gingivitis (ANUG): Smoking is among the etiological factors of ANUG. It has been observed that smoking increases the incidence of ANUG.[35] It has been reported that nicotine-induced contraction of capillaries may cause malnutrition and decrease resistance to infection. It has been suggested that increased steroid hormone levels due to stress or reduced blood flow may suppress host defense mechanisms and exert a selective effect on dental biofilm. This situation may lead to an increase in the number of certain bacteria, such as fusospirochetes, and their high presence in areas with impaired tissue integrity.[36]

Diabetes

Diabetes is a disease caused by disorders in carbohydrate, protein, and fat metabolism, resulting from insufficient production or action of the insulin hormone.[37] Type 1 diabetes occurs due to insulin deficiency resulting from the autoimmune destruction of insulin-producing cells, while type 2 diabetes develops due to the body's resistance to insulin.[38]

The relationship between diabetes and periodontal disease has been reported in the literature since the 1960s, and periodontal disease has been suggested as the sixth major long-term complication of diabetes.[39] The relationship between diabetes and periodontal disease is bidirectional and has been defined as a two-way relationship.[40]

There is a bidirectional relationship between diabetes and periodontal disease. While periodontal infections are more common in individuals with diabetes, it has been reported that periodontal disease negatively affects glycemic control in diabetic patients.[41] There are many reasons for the increased prevalence of periodontal disease in individuals with diabetes. These include altered polymorphonuclear leukocyte function, changes in the immune response, increased oxidation, elevated LDL levels and atherosclerosis, and accumulation of advanced glycation end products in the periodontal ligament and gingiva.[42] Diabetes increases the expression of inflammatory cytokines in the periodontal tissues of individuals.[43] Increased levels of prostaglandin E2 and interleukin-1beta are observed in the gingival crevicular fluid of diabetic individuals.[44] Some studies have reported increased levels of interleukin-1beta, tumor necrosis factor, interleukin-6, interleukin-17, and interleukin-23 in the gingiva of diabetic individuals.[45] Increased expression of inflammatory cytokines leads to increased vascular permeability, inflammatory cell recruitment, reduced osteoprotegerin expression, and increased RANKL expression, thereby contributing to increased destruction.[46]

In a systematic review and meta-analysis conducted by Chavarry et al.,[47] periodontal disease prevalence and severity were found to be higher in 27 out of 49 cross-sectional studies in individuals with diabetes.

In two studies conducted on children aged 6-18 years with type 1 diabetes, the prevalence and severity of attachment loss were reported to be approximately four times higher in these patients.[48]

Some studies have found a correlation between glycated hemoglobin and pH in individuals with diabetes. Additionally, it has been reported that probing depth is greater and attachment loss is higher in individuals with uncontrolled diabetes.[49]

In their study, Nelson et al.[50] reported that the risk of new periodontal disease was 2.6 times higher in individuals with type 2 diabetes compared to healthy individuals. This study also supports the principle of causality, as diabetes developed before periodontal disease.

In a cross-sectional study conducted by Katz et al.[51] on a population of 10,590 individuals, it was reported that

individuals with high blood glucose levels had a higher likelihood of having severe periodontal conditions.

INDIVIDUAL RISK FACTORS

Individual risk factors include risk factors that are specific to the individual and cannot be modified (non-modifiable) and should be limited to this scope. Genetic factors, age, gender, socioeconomic status, and stress are examined as individual risk factors.[11]

Genetics

The predisposition to periodontal disease is based on the interaction between the microbial structure in the area and the host's genetic information. Pathogenic flora can initiate attacks that stimulate the immune response, but this is not always sufficient on its own to cause periodontal destruction. The absence of a consistent correlation between plaque quantity and disease severity, the existence of an individual dose-response curve for each person, and the fact that some individuals develop severe periodontal destruction such as aggressive periodontitis while others do not progress beyond gingivitis or early periodontitis all indicate that the host immune response is more important than bacterial etiology.[52]

There is substantial evidence that genes associated with the host immune response influence the progression of periodontal disease. Variations in different alleles lead to changes in innate immunity, antibody responses in acquired immunity, and nonspecific inflammation. This allows genetics to be considered a risk factor for periodontitis.[53]

Twin studies are conducted to investigate the effect of genetic variation on chronic periodontitis. Identical twins share the same genes because they develop in the same ovary, but fraternal twins do not have the same genetic makeup. Michalowicz et al.,[54] in a study of 110 pairs of twins aged 16 to 70, reported that monozygotic twins showed less variability in clinical attachment levels and mean periodontal pocket depths.

In another study involving 64 pairs of identical twins and 53 pairs of fraternal twins, it was reported that identical twins had a more similar predisposition to periodontitis.[55]

Numerous studies have demonstrated the role of genetics in aggressive periodontitis. Aggressive periodontitis, characterized by rapid attachment loss and severe alveolar bone destruction, is a distinct disease separate from chronic periodontitis. Unlike chronic periodontitis, it may show familial transmission or distribution. Additionally, the role of nutrition, socioeconomic status, environmental factors, and systemic pathologies has been reported in aggressive periodontitis.[56]

Genetic and chromosomal disorders associated with increased susceptibility to periodontal disease include;

- Enzyme and enzyme inhibitor defects
- Acatalasia, congenital erythropoietic porphyria, hypophosphatasia, alpha-1 antitrypsin deficiency
- Leukocyte defects
- Chédiak-Higashi syndrome, glycogen storage disease type Ib,
- Connective tissue disorders
- Ehlers-Danlos syndrome, chromosomal abnormalities of chromosomes VIII and IX
- Trisomy 21 (Down syndrome)
- Leukocyte adhesion defects types I and II, cyclic and chronic neutropenia
- Alopecia, epilepsy, mental subnormality, Papillon-Lefèvre syndrome

Gender

Studies have indicated that gender differences influence the course of many diseases. When factors such as behavioral differences, stress, hormones, and genetics are added, the prognosis of certain diseases may vary according to gender.[58]

Differences in immune responses between genders also contribute to variations in the development of periodontal diseases. Gender hormones and genes associated with the X chromosome are believed to play a key role in these mechanisms.[59]

Sex hormones (estrogen, testosterone, and progesterone) affect immunity. In general, testosterone reduces the immune response, while estrogen increases it.[60] Hormonal changes during puberty, menstruation, pregnancy, and menopause in women, as well as the use of hormonal supplements, have been associated with the development of gum inflammation.[61]

Analyses of NHANES data have estimated that men have approximately 50% higher prevalence of periodontal disease. Compared to women, men had 33% more mild, 28% more moderate, and 180% more severe periodontal disease.[62]

Studies have indicated that women visit the dentist more frequently, place greater importance on oral hygiene, and are more compliant with treatment. Additionally, the National Health and Nutrition Examination Survey conducted in the United States reported that women develop periodontal disease less frequently.[58]

Age

The relationship between periodontal disease and age remains unclear. Early studies indicated that the prevalence and severity of periodontal disease increased with age, suggesting that age could be a predictor of periodontal tissue loss.[63] However, over the years, the notion that periodontal disease is exclusively found in the elderly has been questioned. Studies have indicated that age may not increase susceptibility to the disease but rather reflect the cumulative effect of prolonged exposure to actual risk factors[64] and that periodontal disease can begin in youth/early adulthood.[65]

It has been determined that an individual's level of susceptibility to periodontal disease is more important than age and that the disease develops at an earlier age in individuals with high susceptibility. The effect of age is not the same for periodontal disease and attachment loss. While a significant relationship has been found between increasing age and clinical attachment loss, only a minimal relationship has been observed between age and periodontal disease. After adjusting for common variables such as oral hygiene levels and access to dental services, the effect of age on clinical attachment loss was found to decrease.[66]

In the 1985-1986 U.S. National Survey, where the diagnostic criterion was the presence of 4 mm or more of attachment loss

in one or more areas, the prevalence of the disease was 3.8% in the 25-34 age group and 53.6% in the 55-64 age group.[67]

Another study examining radiographs taken at 10-year intervals found that the annual average alveolar bone loss is between 0.07 and 0.14 mm for individuals aged 25-64, and 0.28 mm for those aged 70.[68]

In a study by Grbić et al.,[69] which included age, dental calculus removal, root surface smoothing, and a seven-month follow-up, a strong association was found with attachment loss of 2.5 mm or more. In the study, 35% of the 30-39 age group showed additional attachment loss, while this rate was 89% in the 60-69 age group.

Socioeconomic Status

When examining data collected in 2014 from 28 European countries, it was observed that unmet oral and dental health needs were higher among low-income groups due to income levels, geographical conditions, and limitations in the healthcare systems they were part of.[70]

Another study examined differences in access to health services between low- and high-income groups resulting from urbanization and reported that people living in rural areas had less access to oral and dental health services across all income groups.[71]

The incidence of gingivitis is higher in people with low socioeconomic status and poor oral hygiene. This situation appears to be related to the frequency of visits to the dentist and dental awareness when compared to socioeconomic status. Socioeconomic indicators are reliable predictors of periodontitis. Differences in access to resources and services that can influence preventive behaviors play a role in the socioeconomic status of periodontal disease. Studies also indicate that education has a greater effect than income in positively influencing the level of periodontal disease.[72]

It has been reported that individuals who brush their teeth twice a day and acquire this habit at an early age develop fewer cavities. The socioeconomic status of families has been reported to be effective in acquiring this habit.[73]

Bostancı et al.,[74] in a study comparing the periodontal status of students attending private and public schools, found that students attending public schools had higher CPITN values. Additionally, Gökalp et al.,[75] in a cross-sectional study involving 7,833 individuals, found that those living in rural areas had more severe periodontal problems.

In a cross-sectional study examining risk factors for periodontal disease, it was found that the prevalence of moderate or severe periodontitis was higher among individuals with lower educational levels. Additionally, the study revealed that the prevalence of moderate or severe periodontitis increased as income levels decreased.[76]

Stress

Stress is among the etiological factors of many inflammatory diseases, including periodontal diseases.[77] Stress is thought to have a negative effect on the formation of the immune response.

During stress, the sympathetic nervous system triggers the release of norepinephrine, which suppresses the immune response.[78] Stress increases the production of glucocorticoid hormones from the adrenal cortex and the synthesis of corticotropin from the pituitary gland; it also reduces the synthesis of pro-inflammatory cytokines.[79]

Hilgert et al.[80] demonstrated that elevated cortisol levels in high-stress conditions are associated with higher levels of periodontal disease.

A study involving 1.426 individuals showed that those who demonstrated excellent coping skills in the face of similar economic difficulties had a lower risk of periodontal disease.[81]

In a small study of 23 individuals examining occupational stress and the progression of periodontal disease, it was reported that increasing age, low socioeconomic status, low job satisfaction, and aggressive behavior significantly increased attachment loss.[82]

Studies have shown that saliva flow rate decreases under stress, and emotional stress affects saliva pH, leading to the development of gum diseases.[83]

The increased incidence of necrotizing ulcerative gingivitis under emotional and physiological stress suggests a connection between the two conditions. Emotional stress affects hormone levels in the bloodstream, which in turn leads to changes in the immune response. It has been reported that the prevalence of periodontal disease increases in stressful situations such as the loss of a close relative, divorce, and economic difficulties.[84]

Stress also affects behavior. In stressful situations, increased smoking, poor oral hygiene, decreased visits to the dentist, and changes in eating habits may be observed. These behaviors can have a negative effect on the periodontal disease process and cause a decrease in the immune response.[85]

RISK INDICATORS FOR PERIODONTAL DISEASE

Periodontal disease risk indicators are risk factors that have been identified in intergroup studies but have not been confirmed by long-term research. These indicators are risk factors that may cause disease or are associated with disease. HIV/AIDS, osteoporosis, and irregular dental checkups fall into this group.[11]

HIV/AIDS

Acquired immune deficiency syndrome (AIDS) is the most severe clinical manifestation of human immunodeficiency virus (HIV) infection. AIDS was first defined in 1981 in the United States as a viral infection that can affect all body systems, has no effective treatment, and can be fatal. AIDS is more common in intravenous drug users, hemophiliacs, patients requiring blood transfusions, bisexual and homosexual men, heterosexual women, and their sexual partners.[86]

In most patients, the first symptom of HIV infection may be oral lesions caused by opportunistic fungal and viral agents. Due to the suppression of the immune system, oral candidiasis is the most common oral condition observed in individuals with AIDS. Additionally, hairy leukoplakia, Kaposi's sarcoma, ulcers, herpes simplex, papilloma, and periodontal diseases may also occur.[87]

More severe necrotizing periodontal diseases often develop in individuals with AIDS. These are caused by spirochetes, fusiform bacilli, gram-negative bacteria, and other microorganisms, as well as malnutrition and poor oral hygiene.

It begins with necrosis of the dental papillae, followed by pain, bleeding, foul breath, fever, and lymphadenopathy. The formation of band-like erythema on the gingival margins by Candida albicans in AIDS patients is defined as marginal gingivitis.[88] HIV infection and AIDS are considered risk factors for periodontal disease.

The prevalence of necrotizing ulcerative periodontitis in AIDS patients and HIV-infected individuals is not precisely known. Some studies indicate that the prevalence of periodontal disease is higher in these individuals compared to others with similar oral hygiene, with prevalence rates ranging between 4% and 5%.[89]

In a prospective study, 114 homosexual and bisexual men were examined over a period of 20 months, and the incidence of active periodontal disease was found to be 6.16 times higher in individuals with CD4 cell counts below 200.[90]

In the first studies conducted on AIDS patients, the prevalence and severity of periodontal disease were higher. However, subsequent studies reported less severe periodontal symptoms. This finding is thought to be related to the successful suppression of the immune system in HIV-positive individuals through high-activity antiretroviral therapy and the continuous development of other medical agents.[91]

Robinson et al.[92] found no difference in the progression of periodontal disease between HIV-positive and HIV-negative individuals in a 12-month follow-up study.

Hofer et al.[93] demonstrated that HIV-positive individuals can be successfully protected in a manner similar to uninfected patients. The specific IgG subclass responses to periodontal pathogens were found to be alike in both HIV-infected and uninfected individuals.

Osteoporosis

Osteoporosis is a metabolic skeletal disease characterized by an increase in bone fragility and fracture risk, as well as a decrease in mineral density, due to deterioration in the microarchitecture of bone tissue.[95]

Non-modifiable risk factors for osteoporosis include age, female gender, family history, previous fractures, race, ethnic origin, menopause, hysterectomy, long-term glucocorticoid therapy, rheumatoid arthritis, and primary/secondary hypogonadism in men. Modifiable risk factors for osteoporosis include inadequate calcium and vitamin D intake, insufficient consumption of fruits and vegetables, excessive protein, sodium, and caffeine intake, eating disorders, insufficient physical activity, frequent falls, and tobacco and alcohol use.[96]

In short-term cross-sectional studies, where most participants were postmenopausal women, it has been reported that women with low bone mineral density had a higher incidence of gum recession, gum inflammation, and attachment loss (von Wowern et al., 1994; Mohammad et al., 1996, 1997; Tezal et al., 2000). However, some studies have not reported such an association (Weyant et al., 1999; Lundström et al., 2001).[97]

Payne et al.[98] reported greater alveolar bone loss in osteoporotic women in their long-term studies. However, Reinhardt et al.[99] reported that serum estradiol levels had no significant effect on attachment loss in their 2-year follow-up studies (**Table**).

Irregular Dental Checks

The issue of whether irregular dental visits are a risk factor for periodontal disease is controversial. One study showed an increased risk of severe periodontitis in patients who did not visit the dentist for three years or more, while another study found no significant difference in attachment and bone loss between those who received dental treatment and those who did not over a six-year period.[11]

Altındal et al.[101] conducted a study between 2016 and 2018 with 1,758 participants, finding a statistically significant association between the diagnosis of periodontal disease and the frequency of dental visits. According to the study results, the majority of healthy individuals reported having previously visited a dentist, while the majority of those diagnosed with periodontitis and gingivitis stated that they had never visited a dentist before.

RISK PREDICTORS/MARKERS FOR THE FUTURE

Signs of future risk prediction/risk signs are associated with an increased risk of disease but do not cause disease. These factors have been identified in intergroup and long-term studies. In this context, factors such as gingival recession and a history of periodontal disease can be examined.[11]

Bleeding During Probing

Dentists associate bleeding during probing with periodontal areas at high risk of future deterioration and use the finding of bleeding during probing as an indication for treatment. However, recent studies have shown that this use should be questioned.[102]

Table. Studies evaluating the relationship between systemic bone loss and periodontitis[100]

Author	Periodontal measurement	Systemic bone measurement	Correlation
Elders et al.	Alveolar bone height	Dual photon absorptiometry, L2-L4, metacarpal thickness	No
Klemetti et al.	Index of periodontal treatment needs in the community	Dual-energy X-Ray absorptiometry of the spine and hip	No
Hildebolt et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine and hip	No
Mohammad et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine and hip	Yes
Lundstrom et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry of the hip	No
Mohammad et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine	Yes
Taguchi et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry of the spine and femoral neck	No
Ishii et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry of the femoral neck	No
Brennan et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine, hip, and forearm	Yes
Payne et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry	Yes
Yoshihara et al.	Clinical detachment	Dual-energy X-Ray absorptiometry	No

Gbric and Lamster,[103] in their 6-month study of 75 patients, found that 68% of areas showing active disease had bleeding, and 57% of areas without active disease also had bleeding.

Kaldahl et al.[104] conducted a study to determine active disease areas, examining patients who had undergone maintenance therapy for two years. Their findings concluded that the presence of bleeding during probing does not predict future attachment loss.

Previous Periodontal Disease

Many clinical studies have shown that individuals who have had periodontal disease in the past are at higher risk of developing the disease again in the future compared to those who have not had it.[105] The most common situations in which disease recurrence occurs after treatment include poor oral hygiene, smoking, systemic diseases such as diabetes, and non-compliance with treatment.[106] Adherence to regular supportive periodontal therapy (SPT) plays an important role in reducing disease recurrence and tooth loss.[107]

In a meta-analysis conducted to evaluate the effectiveness of different methods in assessing disease recurrence and managing recurrent disease in individuals who had undergone long-term supportive periodontal care (SPC) and had previously received periodontitis treatment, the rate of patients experiencing multiple 2 mm attachment loss was found to be 24.8%.[108]

In a study conducted in Iran involving 50 patients (29 men and 21 women) who had completed periodontal treatment but did not adhere to the maintenance phase, a significant association was found between the initial diagnosis and the recurrence rate of periodontitis (p=0.017). Additionally, a significant relationship was found between the number of years post-treatment and periodontitis recurrence (p=0.027, r=0.353).[109]

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Referee Evaluation Process

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The authors have no conflicts of interest to declare.

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All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

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Treatment of Miller class 3 gingival recession with free gingival graft and creeping attachment phenomenon: 2 case reports; 2-year follow-up

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ABSTRACT

Periodontal plastic surgical procedures applied to regulate the gingival-mucosa relations that complicate periodontal diseases and affect the success of periodontal treatment are referred to as mucogingival surgery. It is known that complete root surface closure can be achieved in mucogingival surgical procedures to be applied in patients without interproximal bone loss. However, recent studies suggest that patients with bone loss can also be treated successfully. The movement of the marginal edge of the gingiva towards the coronal margin after mucogingival surgery is defined as 'creeping attachment'. This case report aims to emphasize the importance of creeping attachment after free gingival grafting in patients with Miller class 3 gingival recession. Intraoral and radiologic examinations of 2 systemically healthy, non-smoking female patients aged 27 and 28 years showed mild to moderate periodontal bone loss and were diagnosed as stage 1 periodontitis. Patients with frenulum recession and gingival recession in the mandibular incisor region had Miller class 3 recession. Free gingival graft was applied to the patients and the changes in the graft and gingival recession area and creeping attachment were observed for 2 years. After free gingival grafting, in addition to the initial postoperative root surface closure in patients with Miller class 3 gingival recession, it was observed that the size of the gingival recession decreased or the root surface closure rate increased with creeping attachment over time.

Keywords: Free gingival graft, creeping attachment, mucogingival surgery, gingival recession

INTRODUCTION

Gingival recession is a common condition encountered in the society and is defined as the displacement of the gingival margin towards the apical part of the enamel-cementum junction.[1] It has been shown in studies that gingival recession increases with age, especially in young people, recession is seen mostly on buccal surfaces and affects interproximal areas in advanced ages.[2]

Gingival recession may occur for many reasons. One of the most important causes is plaque-related inflammation of the gingiva. Incomplete oral hygiene due to insufficient amount of keratinized gingiva, tooth movements caused by forces applied in orthodontic treatments, abnormal muscle and frenulum connections and occlusal trauma may also cause gingival recession.[3] Tooth brushing habits, bristle thickness, brushing technique, brushing force and frequency have been associated with gingival recession, especially on buccal surfaces.[4,5]

Along with aesthetic problems, gingival recession may cause dentin sensitivity, root caries, cervical lesions, exacerbation of periodontal diseases due to plaque accumulation and eventually tooth loss. These conditions can be treated with mucogingival surgery.[6,7] Today, the main goal of mucogingival surgery is the correction of defects related to gingival recession and very successful results are obtained in the treatment of these problems with free gingival graft applications.[7]

Free gingival grafts, which were previously used only to increase the width of keratinized gingiva, are among the mucogingival surgical procedures that have been successfully used to cover the root surface in gingival recession, to eliminate abnormal frenulum and muscle attachments, and to provide patients with an appropriate gingival-mucosa relationship where they can provide effective oral hygiene.[8] However, it is recommended to be used in the premolar region and lower anterior region because the color and texture characteristics are not fully compatible.[1,9]

In 1968, Sullivan and Atkins outlined the surgical principles of free gingival graft application in isolated gingival recession. By placing the free gingival graft on the avascular bare root surface, a permanent bridge is obtained and primary closure of the root surface is achieved.[10] In addition to this primary closure, Goldman described the secondary root surface closure that occurs over time as creeping attachment.[11] This passive covering process that occurs after surgery has been

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described by many authors as migration of the gingiva towards the coranal.[12,13]

In this case report, dimensional change and creeping attachment were observed within two years after the primary closure of the root surface with free gingival graft treatment applied to different patients, and the secondary root surface closure that occurs over time was demonstrated. It has been shown that favorable results can be obtained with free gingival grafting in patients with interproximal periodontal bone loss. At the same time, it is aimed to demonstrate the conditions that can be achieved by waiting before deciding to perform a second surgical procedure in root surfaces that are not completely closed after the surgical procedure.

CASE 1

A systemically healthy 27-year-old woman was admitted to Dicle University, Faculty of Dentistry, Department of Periodontology with bleeding, pain and gingival recession. She was a non-smoker and brushed her teeth once a day. Bleeding on probing, extensive calculus accumulation and minimal bone loss radiologically not exceeding the coronal part of the root were observed. The patient had stage 1 periodontitis and Cairo type 2 (Miller class 3) gingival recession with abnormal frenylum attachment and shallow vestibular sulcus depth in the mandibular incisors. Phase 1 periodontal treatment (scaling, root planing and oral hygiene instructions) was performed. After the gingival index and plaque index scores were zeroed, a free gingival graft operation was planned (**Figure 1a**). Informed consent was obtained for the operation. In teeth 31 and 41, root surface smoothing was performed using gracey curettes (Hu-Friedy® Gracey curette, Chicaho, USA). The operation was continued with intrasulcular incisions made at the gingival sulcus of the teeth and horizontal incisions to coincide with the enamel-cementum border. Afterwards, vertical incisions were made distal to both teeth starting from the end of the horizontal incisions and extending to 3-4 mm apical to the extraction defects. With sharp dissection, the frenylum and muscle attachments were completely eliminated and an immobilized recipient bed was created. The palatinal palate region of the patient was used as the donor site and a 2 mm thick free gingival graft was obtained under local anesthesia in accordance with the shape of the recipient site and the palate was sutured with 3/0 silk suture after controlling bleeding. The free gingival graft was immobilized in the recipient bed using 5/0 non-resorbable polyamide suture (**Figure 1b**).

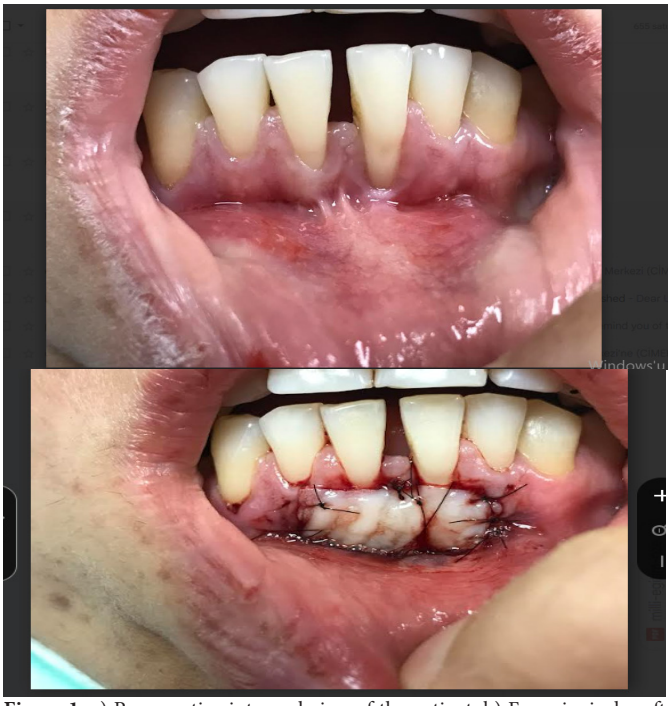

Figure 1. a) Preoperative intraoral view of the patient, b) Free gingival graft application and suturing

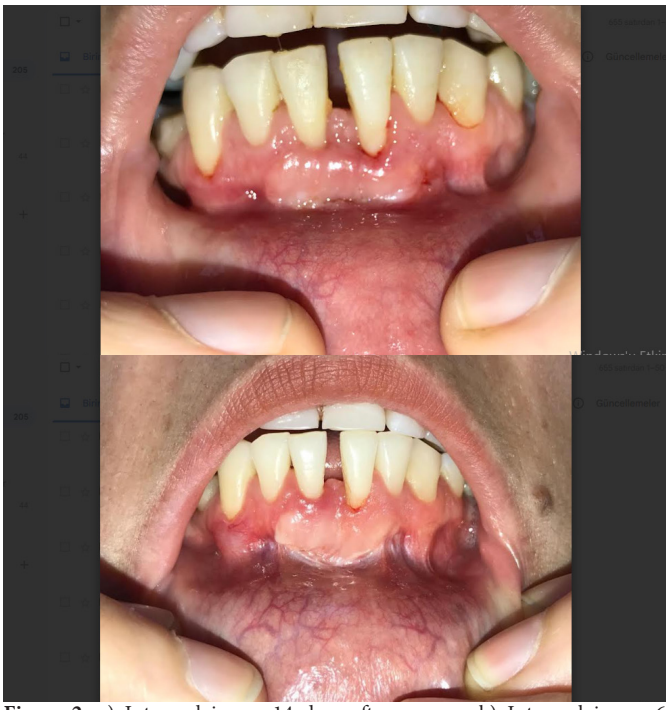

Figure 2. a) Intraoral image 14 days after surgery, b) Intraoral image 6 months after the operation

The patient was given a post-op order. Analgesics, anti-inflammatories and antibiotics were prescribed for 1 week (Eto-dolac 400 mg tablet and Amoxicillin/Clavulanic acid 625 mg film tablet). She was asked to continue 0.12% chlorhexidine gluconate mouthwash for 15 days, to protect the area from any trauma and not to brush the operation area for 15 days. After 14 days, sutures were removed (**Figure 2a**). Afterwards, the patient was called for follow-up visits at 6, 12 and 24 months and the changes in the free gingival graft were observed (**Figure 2b, 3a, 3b**).

At the time of suture removal, it was seen that the gingival margin was not in the desired area in the collar region in both teeth and there was a gingival recession of 1 mm in tooth number 41 and 2.5 mm in tooth number 31. At the 6-month to 1-year controls, a complete root surface closure was obtained in tooth number 41 with creeping attachment in the tissue that was left to heal passively, while a 1.5 mm closure was achieved in tooth number 31. Between the 1st and 2nd year, creeping attachment was slower and less frequent. The final result was satisfactory for the patient and the patient's complaints were completely resolved.

CASE 2

A 28-year-old systemically healthy, non-smoking female patient was admitted to our clinic with complaints of bleeding, pain, gingival recession and tenderness. Abnormal frenulum recession in the region of the mandibular incisors, bone loss in the interdental regions and Cairo type 2, miller class 3

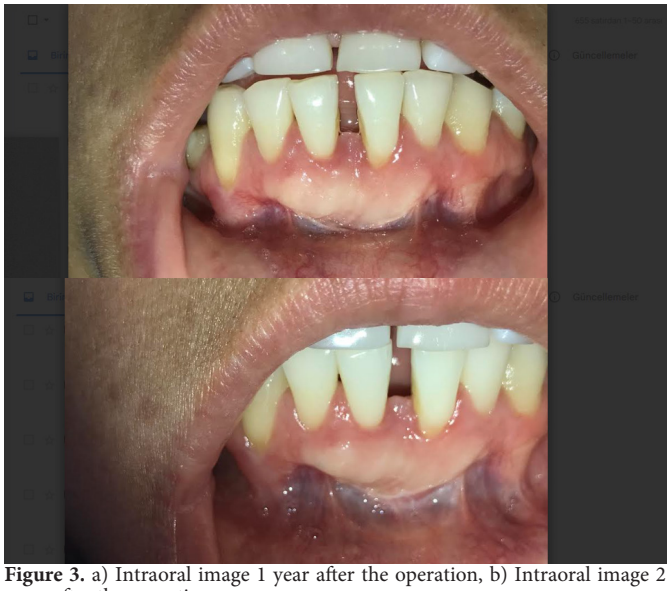

Figure 3. a) Intraoral image 1 year after the operation, b) Intraoral image 2 years after the operation

of the gums were detected. Intraoral and radiologic examination revealed Stage 1 periodontitis with bone loss not exceeding the coronal triangle of the root. Detethering, subgingival curettage, root surface smoothing were performed and the patient was followed up until the intraoral scores became appropriate and oral hygiene was expected to stabilize (**Figure 4**).

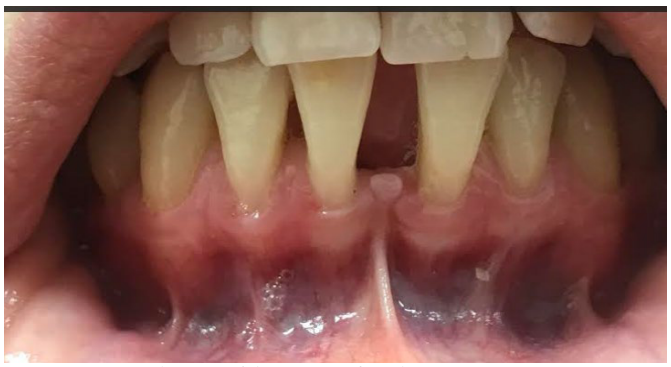

Figure 4. Intraoral image of the patient after phase 1 treatment

After the initial treatment, it was decided to apply free gingival graft to the lower incisor area where the patient's complaints were maximal. The first incision was made parallel to the mucogingival line, leaving free gingiva in the collar region of the teeth. In the recipient bed, the frenulum was completely neutralized and a suitable immobilized bed was created (**Figure 5a**).

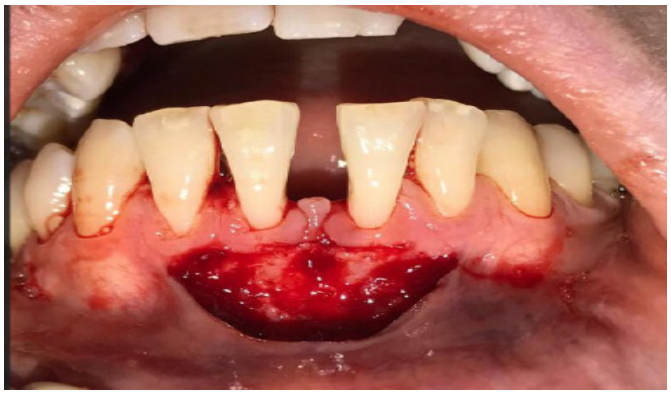

Figure 5. a) Preparation of the recipient bed, b) Suturing the free gingival graft to the recipient bed

The free gingival graft taken from the palate was fixed to the area using simple sutures, horizontal matrix sutures and pressure sutures. Pressure was applied on the graft with a wet spacer to prevent dead space under the graft (**Figure 5b**). The patient was prescribed analgesic, anti-inflammatory, antibiotic and chlorhexidine mouthwash. She was asked not to brush the area for 2 weeks and to protect it from trauma. After 14 days, there was no necrosis when the sutures were removed.

After 6 months, creeping attachment increased, however, the free gingival graft appeared quite protruding on the tissue and its form was not at the desired level **Figure 6a, 6b**). At the end of the 2nd year, it was seen that the graft compliance increased and root coverage was maximized (**Figure 7a, 7b**).

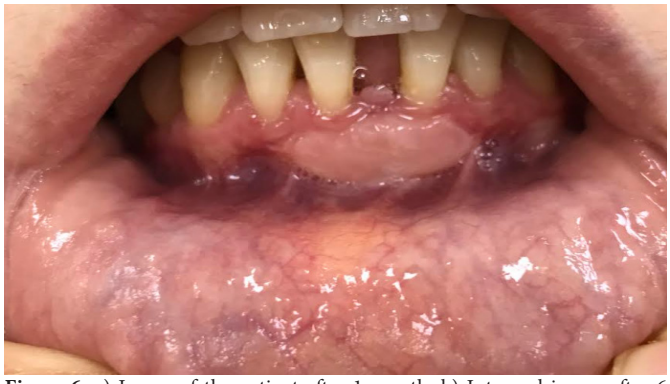

Figure 6. a) Image of the patient after 1 month, b) Intraoral image after 6 months

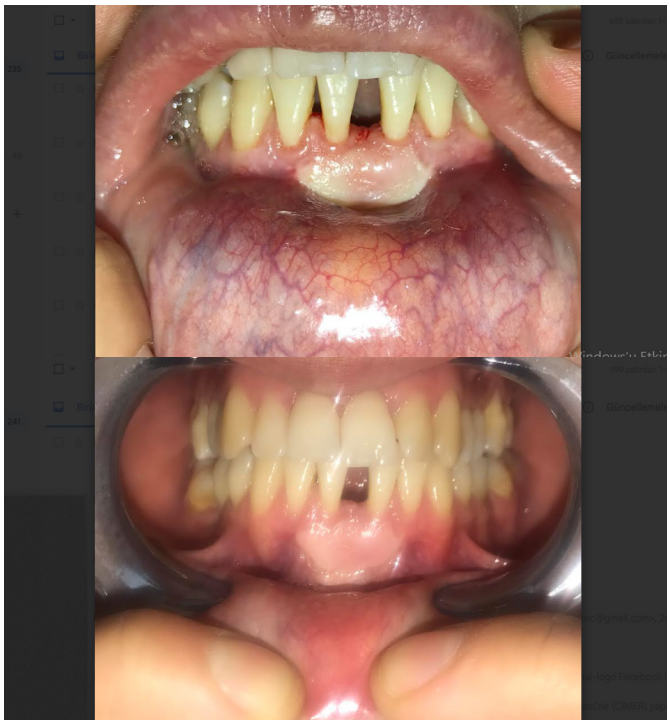

Figure 7. a) Intraoral image of the patient after 1 year, b) Intraoral image after 2 years

DISCUSSION

Free gingival grafts are the most effective mucogingival surgical procedure used to increase the width of insufficiently keratinized gingiva and are frequently preferred to cover the root surface when there is an appropriate indication.[14,15]

It is known that complete root surface closure can be obtained in mucogingival surgical procedures to be performed

especially in patients without bone loss at the interfaces such as Miller class 1 and 2.[7,16] There are not many publications reporting success rates after surgeries performed in Miller class III gingival recessions in which a partial root surface closure is predicted due to the presence of attachment loss and periodontal bone loss in the interdental area.[17] However, complete root surface coverage has been achieved in Miller class 3 cases by applying different surgical techniques. Recent studies suggest that patients with interproximal bone loss can also be treated successfully.[15,17,18] For this, the width and height of the papilla in the interproximal area, limited interproximal bone loss, and an appropriate graft thickness are extremely important, Miller stated in a recent article.[19] Both of our patients had interproximal bone loss and Miller class III gingival recession. As stated in previous studies, despite the presence of bone loss, one of our patients had complete root surface coverage and the other had near complete root surface coverage.

The primary aim in free gingival graft applications is not to close the root surface; however, in narrow recessions with a width of less than 3'mm, root surface closure with creeping attachment can be realized both after surgery and over time.[8] Studies have shown different results regarding when creeping attachment starts after surgery and how long it continues. Many studies have argued that creeping attachment starts between 2 weeks and 6 months.[20,21] In addition to the studies suggesting that the end period is between 1 and 5 years, there are also studies suggesting that it continues for a longer period.[22] Therefore, there is no definite interval for the ideal follow-up period of creeping attachment and this period is uncertain.[10] Although it is not possible to say precisely when creeping attachment started the the coverage of the root surface increased significantly during the 2 years of observation.

CONCLUSION

In this case report, it was demonstrated that positive results can be obtained with free gingival grafting in patients with Miller class III gingival recession. Within 2 years, the depth of recession decreased, the amount of keratinized tissue increased and successful results were obtained in covering the root surface with creeping attachment.

ETHICAL DECLARATIONS

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Journal of Dental Sciences and Education

Case Report

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Anterior diastema closure with direct composite resin: three case reports

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ABSTRACT

Anterior maxillary and mandibular diastemas are common aesthetic concerns reported by patients. The treatment of these diastemas involves various approaches, including surgical interventions, orthodontic applications, restorative procedures, or a combination of these methods. Achieving successful outcomes requires careful consideration of the appropriate technique and material selection, along with time constraints, physical and psychological factors, and economic limitations. In diastema cases, direct composite resins offer both clinicians and patients the ability to effectively manage these challenges, facilitating the creation of a natural smile. This case report presents the treatment of three individuals with anterior diastemas. The diastemas were successfully managed in a single session using direct composite resin restorations without the need for tooth preparation.

Keywords: Diastema closure, direct anterior restorations, adhesion, aesthetic

INTRODUCTION

Diastema is a dental anomaly characterized by gaps in the interdental region and the absence of contact points. It is often considered an aesthetic or malocclusion issue, making its closure a common practice in cosmetic dentistry.[1,2] However, not all diastemas require correction. Decisions regarding diastema closure depend on various factors, including the etiology of the problem, the patient's economic status, time, and the patient's preferences. Several treatment methods can be used for diastema closure. Explaining different treatment options to the patient is crucial for achieving a successful outcome and ensuring the patient's consent and cooperation. Depending on the etiological factors, different treatments such as surgery, orthodontics, restorative procedures, or a combination of these may be applied.[1,3,4] By considering the patient's needs, requests, and expectations during the treatment planning process, patient satisfaction with the treatment outcomes can be ensured.[5]

The factors that can cause diastemas are multifactorial and can be listed as follows:[4,6]

- Genetic or racial characteristics
- Labial frenulum
- Imbalanced muscle function
- Dental and jaw malocclusions
- Dental pathologies (microdontia, mesiodens, conical lateral incisors, lateral incisor agenesis, cysts in the midline region)
- Harmful habits (thumb sucking, tongue thrusting, and lip sucking)
- Anomalies in the shape, size, and number of teeth
- Tooth loss
- Physiological or pathological tooth movements
- Maxillary incisor proclusion

Orthodontic treatments are generally recommended for closing diastemas, but in some patients, diastemas may persist after treatment due to changes in dental proportions. Additionally, orthodontic treatments can be expensive and time-consuming. Faster treatments are associated with restorative procedures such as ceramic veneers and composites.[3,7]

Ceramics, despite their aesthetic properties, are generally more expensive than composite resins because they require multiple sessions and laboratory stages. On the other hand, direct composite restorations can be completed in a single session, are easily repaired, and possess both functional and aesthetic properties, making them a preferred choice.[8] Additionally, the rapid advancement of adhesive technology has made composites applicable with minimal or non-invasive approaches, requiring little or no preparation, further enhancing their appeal.[3,7,9]

CASE PRESENTATIONS

Three patients, two of whom were female, presented to the Restorative Dentistry Clinic of the Faculty of Dentistry at Dicle University with complaints of diastema in their anterior teeth. A detailed medical history evaluation revealed no systemic disorders in the patients. Clinical oral examination and radiographic evaluations revealed no evidence of dental caries, and the periodontal tissues were found to be healthy.

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The patients were provided with detailed information about the options for closing the diastema. The advantages and disadvantages of composite restorations were explained, and the patients' socioeconomic status, age, preferences, and expectations were considered. As a result, it was decided to treat the teeth with direct composite restorations as a more protective, economical, aesthetic, and quick option. Detailed information was provided before the interventional procedures, and informed consent forms were obtained from the patients.

Before isolation, color selection was made using the manufacturer's color scale in daylight. The total etching method was preferred to achieve high bond strength. A 37% phosphoric acid (Scotchbond; 3M ESPE) was applied to the surfaces of the teeth to be restored for 30 seconds, followed by rinsing for 30 seconds and drying.

Transparent tape was placed on the mesial and distal surfaces of the teeth to cover the gingival sulcus. Then, a bonding agent (Tokuyama Universal Bond, Japan) was applied using a brush. Light air was applied to create a thin layer of bonding material. The material was polymerized using an LED light source (LED E Plus polymerization filling device) for 10 seconds. Composite resin material [A2, A3 Tokuyama Estelite Σ Quick (Tokyo, Japan)] was placed on the prepared tooth surfaces using a layering technique and exposed to LED light for 20 seconds. After polymerization, the transparent tape was removed and occlusion was checked. Excess material around the cavity margins was removed using micro-granulated flame-tipped burs. The contours of the gingival margins were adjusted, and the finishing process was completed using discs and polishing rubber cups. Patients were informed about the precautions to be taken after treatment and advised to return for follow-up visits at six-month and one-year intervals.

CASE 1

A 35-year-old female patient presented to our clinic with discomfort caused by diastema in her maxillary anterior teeth (**Figure 1**).

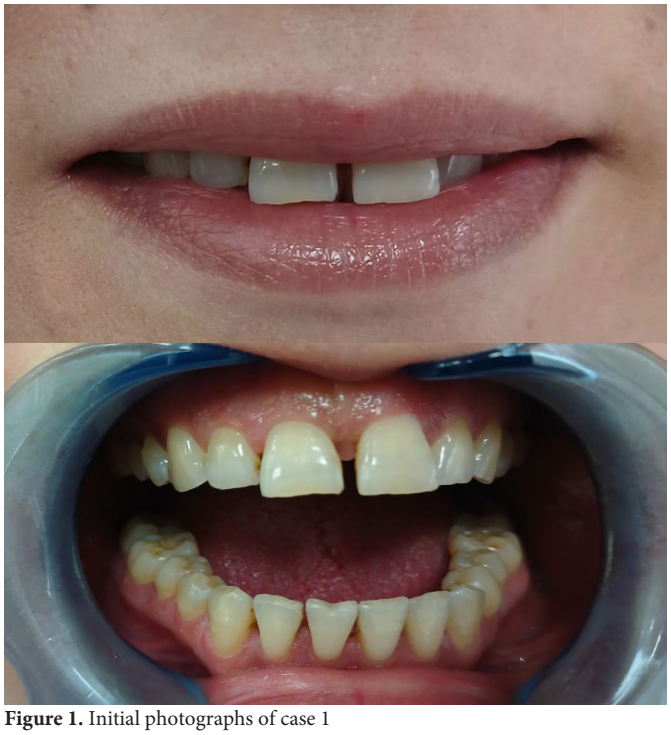

Figure 1. Initial photographs of case 1

As treatment, it was decided to apply the direct composite resin technique, a minimally invasive method, to the teeth as described above. After polishing the teeth, the total acid etching procedure was performed without any preparation. Following the bonding process, the teeth were shaped to match their natural form using composite resin [A2 Tokuyama Estelite Σ Quick (Tokyo, Japan)]. As a result, a smile aesthetic that met the patient's expectations was achieved (**Figure 2**).

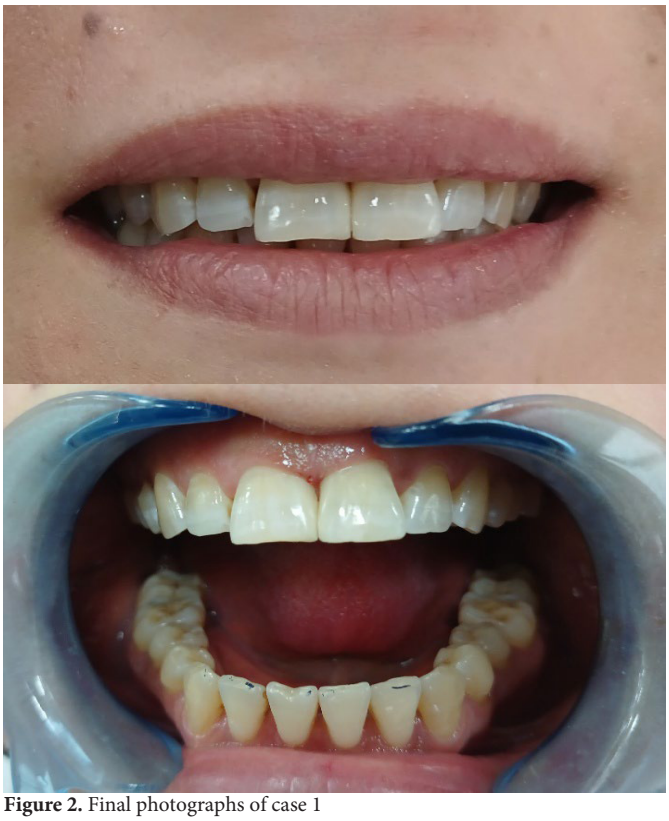

Figure 2. Final photographs of case 1

CASE 2

A 34-year-old male patient presented to our clinic with aesthetic complaints due to diastemas in his maxillary anterior teeth. Examination revealed diastemas between the central and lateral teeth (**Figure 3**). The patient's periodontal health was found to be good, with normal vertical and horizontal occlusion.

As in Case 1, treatment options were explained to the patient, and a joint decision was made with the patient to apply restorative treatment using the direct composite resin technique in a way that would cause the least damage to the teeth. Following polishing, the total acid etching procedure was performed without any additional preparation. After bonding, the teeth were shaped into their natural form using composite resin [A2 Tokuyama Estelite Σ Quick (Tokyo, Japan)], resulting in a satisfactory aesthetic smile for the patient (**Figure 4**).

CASE 3

A 30-year-old female patient presented to our clinic with aesthetic complaints due to a diastema in the mandibular anterior region (**Figure 5**). After the treatments done in cases 1 and 2, we finished the restoration using composite resin material [A3 Tokuyama Estelite Σ Quick (Tokyo, Japan)] (**Figure 6**).

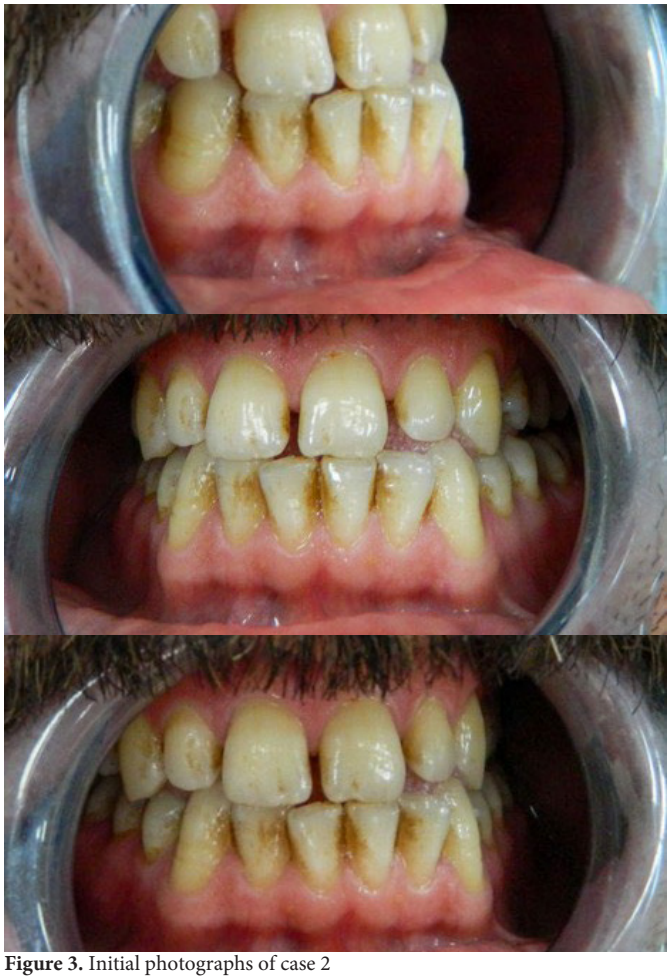

Figure 3. Initial photographs of case 2

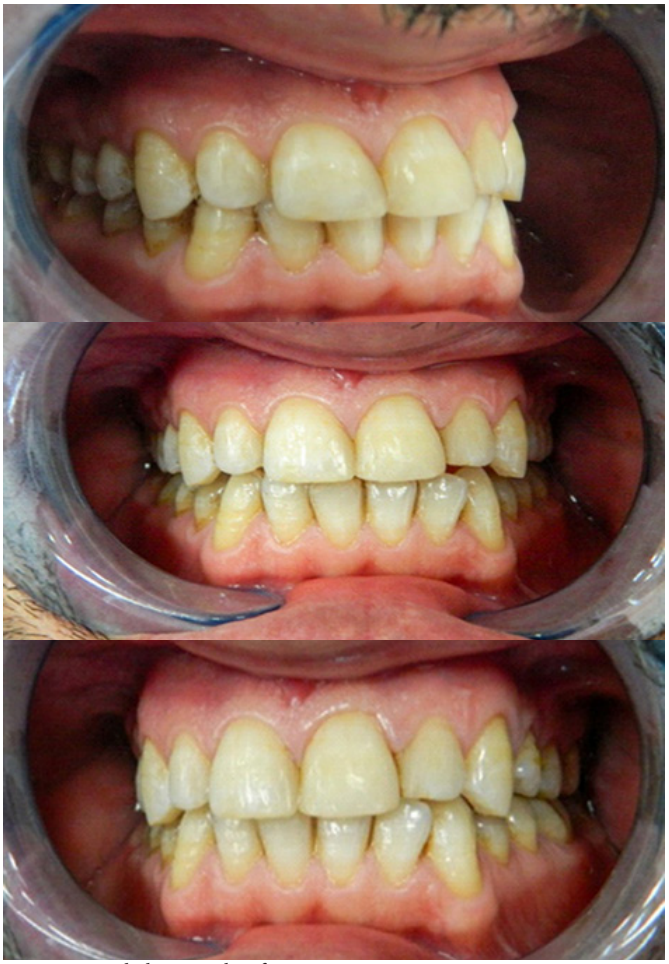

Figure 4. Final photographs of case 2

CASE 3

A 30-year-old female patient presented to our clinic with aesthetic complaints due to a diastema in the mandibular anterior region (**Figure 5**). After the treatments done in cases 1 and 2, we finished the restoration using composite resin material [A3 Tokuyama Estelite Σ Quick (Tokyo, Japan)] (**Figure 6**).

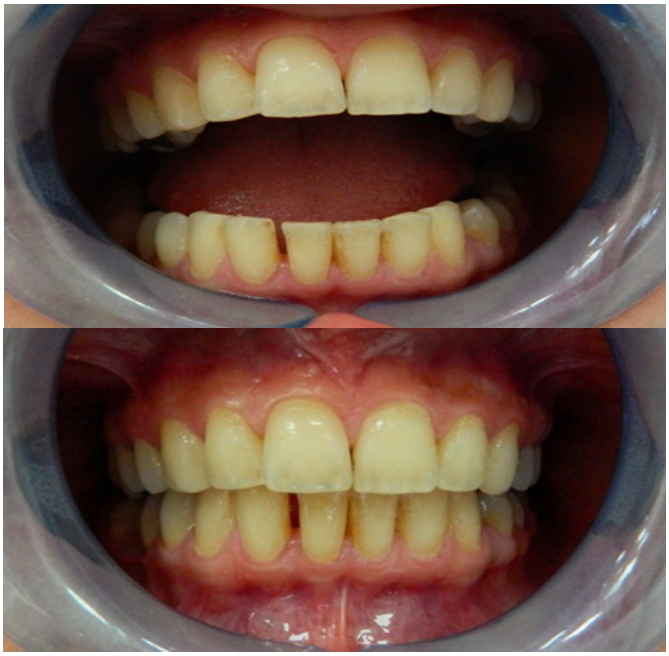

Figure 5. Initial photographs of case 3

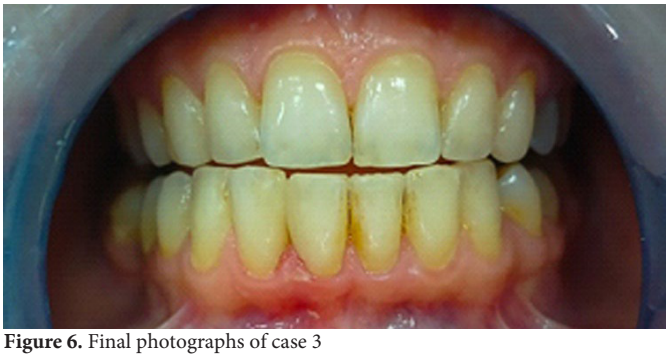

Figure 6. Final photographs of case 3

DISCUSSION

The most suitable options recommended for closing diastemas include orthodontics, operative dentistry, and prosthetic applications. Orthodontic treatments require the use of fixed appliances, which entails a more complex, lengthy, and costly treatment process. Prosthetic dentistry, on the other hand, involves indirect and more invasive procedures that require laboratory participation.[10] At the same time, prosthetic methods preferred to correct aesthetic problems, especially to correct color, shape, and position defects in anterior teeth, may cause excessive tooth reduction, which can damage tooth structure and periodontium.[2]

Direct composite restorations offer numerous advantages, including the ability to be applied in a single session, no need for additional laboratory procedures, and features not available in other potential treatment options such as ceramic veneers and orthodontic treatment. Additionally, their ability to be repaired more easily and at a lower cost compared to

porcelain alternatives in the event of unexpected fracture, and the absence of time-consuming repair or reconstruction procedures, are significant advantages.[6] However, direct composite restorations have some disadvantages compared to indirect porcelain alternatives. Most composite materials are not ideally suited for high-stress areas due to their lower fracture resistance and lower pressure resistance in certain clinical situations.[11]

The presence of uncontrolled parafunctional forces, such as bruxism, Class III edge-to-edge occlusion, or nail-biting, can potentially compromise the longevity of direct composite restorations. Additionally, the color stability of direct composite restorations is not as inert as polished ceramics; however, the outcome depends on the quality of finishing and polishing procedures and can be prevented with proper controls.

Despite the disadvantages of direct composite restorations, advancing adhesive techniques and higher-quality composite materials are providing dentists with the opportunity to create more protective, functional, aesthetic, economical, and long-lasting restorations in a very short time.[12]

The presence of diastemas is one of the important factors that can lead to interdental papilla deficiency.[13] One of the main challenges encountered in restorative procedures aimed at closing diastemas is the inability to prevent the formation of wide gingival embrasures, often referred to as the "black triangle."[14]

Studies have indicated that the distance between the contact point created during diastema closure treatment and the height of the alveolar bone crest directly affects interdental papilla formation. It has been reported that papilla formation was observed in nearly all cases when the distance was 5.0 mm or shorter. However, when this distance increases to 6.0 mm, the papilla formation rate drops to 56%, and when it reaches 7.0 mm, it decreases to 27%. These findings emphasize the importance of positioning the contact point in a manner that supports papilla development during restorative procedures.[15]

Direct composite restorations are a preferred treatment option for both dentists and patients due to their minimal invasiveness, quick application, and aesthetic success.[16] A successful treatment approach requires consideration of numerous limiting factors, including time, individual characteristics, psychological status, and economic conditions. Direct composite resins used in diastema cases allow for the controlled management of these limitations while enabling the achievement of natural smile aesthetics.

Additionally, if vital whitening treatment has been applied to the patient prior to diastema closure to achieve an effective smile aesthetic, it is recommended that the composite resin application be performed two weeks after the whitening procedure to ensure optimal bonding. Studies have shown that the bonding strength of restorations decreases by 25-60% following tooth whitening procedures.[17]

Limitations

As a limitation, the absence of long-term clinical follow-up restricts the ability to evaluate restoration longevity and color stability over time.

CONCLUSION

Diastema is a condition characterized by interdental spaces commonly seen in the front teeth, which can affect an individual's aesthetic appearance and oral functions. Various treatment options are available for the effective management of this condition, and the advantages and limitations of each approach should be carefully evaluated. The key to achieving clinical success lies in establishing the most appropriate treatment plan tailored to the patient, alongside an accurate diagnosis. In this context, personalized treatment approaches play a significant role in meeting aesthetic expectations while also facilitating functional improvements.

In cases where esthetic demand, limited time, and a minimally invasive approach are priorities, direct composite restoration can be considered a first-line treatment option for diastema closure.

ETHICAL DECLARATIONS

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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